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Editorial

ON DAFFYDILS

Fair Daffydil, we weep to see
You haste away so soon;
The Caput says your time is done
And ours will come in June.
With apologics to Robert Herrick.

To those who are not particularly versed in the science of Horticulture, one should perhaps explain that on the grounds of the University of Toronto are nurtured an odd half dozen species of exquisite annual flowers, each the pride of a Faculty. These were propagated here in Toronto and appear in no other botanical gardens in the world, for the simple reason that the soil must be extremely rich and such fertility is not to be found elsewhere. The most beautiful of these rare flowers is the Daffydil, which was first produced by the Medical Faculty in 1903 and planted on very fertile soil at the south-east end of the campus. The seeds are sown in December under the direction of an experienced horticultural-

ist and assisted by an able staff, the beautiful flowers blossom in late February or early March, are harvested and displayed at the annual Flower Festival in Hart House. Of late, the fame of this rare species has become so universally acknowledged that of necessity the Festival has been extended to three nights in order that all flower lovers might enjoy the blossoms and follow their improvement from year to year.

One might judge that because the Daffydil appears so early in the Spring, it is a hardy flower; but unfortunately such is not the case for I have stated that the soil must be extremely rich and, in spite of expert attention, a blight not infrequently appears on parts of the crop which passes unnoticed until blossom time, when it is only too apparent. It is the belief of a few who have attended the Flower Festival for some years that the blight has recently increased to the point of threatening the popularity of the display. This opinion was conveyed to the horticulturalists who had been carefully selected to plant and nurture the 1940 crop of Daffydils, and every precaution was taken to rid the species of this harmful agent. After much study and research a protein derivative was discovered which has been named "Purine" (the chemical nature and effects of which will be described in the next issue of the annual Horticultural Magazine, *Epistaxis*). The chief horticulturalist (who has had conspicuous success in growing Daffydils during the past six years and who prefers to remain anonymous) personally supervised the spraying of the crop soon after seeding in December, and at repeated intervals until the buds commenced to blossom forth, when everyone was gratified to see that the flowers indeed were free of blight and there was every prospect for the most successful Flower Festival in the history of the University.

Ten days before harvesting time, a cool breeze arose from the south-west end of the campus and so rapidly increased in intensity and decreased in temperature that in spite of the attempts of 700 anxious Daffydil admirers to shield the crop from the frigid blast, the delicate blossoms were soon rendered unfit for the anticipated display which was promptly cancelled by the confused attendants who had worked so admirably to improve and perpetuate this rare flower.

We who this year leave the University, pass on to others the responsibility of reviving and improving the species, and express the hope that in 1941 a crop of Daffydils will rear their heads, confident of their ability to withstand the elements.

THE ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA

In 1939, eight undergraduates of the Toronto School of Medicine obtained their Junior Canadian Fellowships, which will entitle them to try the Senior examinations in either Medicine or Surgery after spending three years in the study and practice of the profession.

In this issue of the JOURNAL is published an announcement of the 1940 examinations, and it is to be noted that only one set is scheduled. Copies of the written examinations to date, with information on refresher courses and fees, may be obtained from the Honorary Secretary, Dr. W. S. Lyman.

The fact that the larger Canadian hospitals state on their interne application forms their preference for graduates who have passed the primary examinations, demonstrates the importance which this degree is coming to assume.

HAEMORRHOIDS

THEIR SIGNIFICANCE AND TREATMENT

By J. T. DANIS, M.B., F.R.C.S., Edin.
Department of Surgery, University of Toronto

Haemorrhoids are varicose dilatations involving one or more radicals of the haemorrhoidal veins. The term "*haemorrhoid*" is not a suitable one but has been accepted through the years as denoting pathological changes in the venous drainage system of the lower rectum and anal canal. The condition is so common after the age of forty, that a clear conception of their development and treatment should be of interest to the student and graduate alike.

As the situation of haemorrhoidal tissue is fairly constant and always involves the terminal branches of the haemorrhoidal arteries and the associated veins it is obvious that in order to appreciate the frequent pathological changes occurring in this region a detailed knowledge of the arterial and venous systems of these parts is essential.

The Arterial Supply of the Rectum and Anal Canal

The chief arterial supply of the rectum and upper part of the anal canal is from the *superior haemorrhoidal artery*, a direct continuation of the inferior mesenteric artery. It descends into the pelvis in the root of the pelvic meso-colon and opposite the third sacral vertebra (the beginning of the rectum) it divides into two main branches, the right and left superior haemorrhoidal arteries, to be distributed to the corresponding halves of the rectum and upper part of the anal canal. Having reached the middle of the rectum they pierce the muscular coat to reach the submucous spaces. From this point the manner of distribution of these two arteries differs somewhat.

Let us consider first the **right superior haemorrhoidal artery.** It divides into two primary branches; an anterior and a posterior primary branch. In order to facilitate subsequent descriptions I would refer you to Fig. 1 in which the upper part of the anal canal has been divided into four quadrants by vertical and horizontal lines. In this diagram the patient is in the lithotomy position, the top of the diagram being anterior, the reader's left being the patient's right, etc. Between the two circles lies the lower half inch of the anal canal lined by squamous epithelium and never the seat of haemorrhoids because the subepithelial tissue is scanty and so firmly adherent to the squamous layer that the few veins present are never capable of distention. The middle circle represents the pectinate line, line of anal papillae, or the junction between the squamous epithelium below and the columnar epithelium above. The mucosa above the pectinate line is disposed in longitudinal columns covering the similarly disposed veins and arteries. These are the Columns of Morgagni, and there are from six to ten of them. The inner circle represents the upper limit of the anal canal—where the rectum perforates the pelvic diaphragm. We have traced in order, down to this point, the superior haemorrhoidal artery, its two main branches, the right and left superior haemorrhoidal arteries, the right dividing into two primary branches, anterior and posterior. The anterior primary branch runs a straight course (as all primary branches do) down the right anterior quadrant of the lower rectum and finally breaks up into a meshwork of small vessels in the submucosa of a Column of Morgagni. This last mentioned artery does not give off any secondary branch during its course along the rectal wall. When the veins accompanying this branch become tortuous, dilated and thickened, they give rise to the *right anterior primary haemorrhoid* which always remains discrete, is constant in its position and never has any

connection with neighbouring haemorrhoids (Fig. 2). *The posterior primary branch* passes downwards into the right posterior quadrant to terminate in a meshwork in a corresponding Column of Morgagni. During its course it gives off *two secondary branches*, an *anterior* and a *posterior*. The former terminates in a Column of Morgagni at a point separating the right anterior from the right posterior quadrant, the latter passes downwards to end in a similar manner in the middle line posteriorly. The

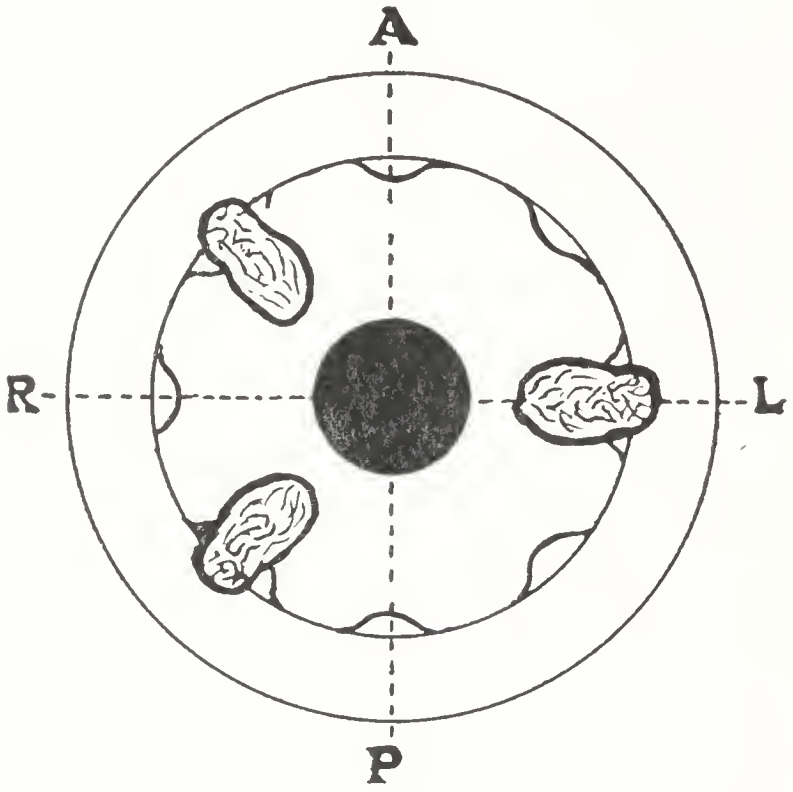

FIG. 1.—Diagram showing the three primary internal piles—right anterior, right posterior and left.

veins accompanying the *posterior primary branch* give rise to the *right posterior primary haemorrhoid*, those accompanying the *secondary branches* give rise to the *right* and to the *posterior secondary haemorrhoids* respectively. These secondary haemorrhoids are much smaller than the primary haemorrhoids and are frequently absent. So much for the right side of the rectum; let us now consider the left side.

The left superior haemorrhoidal artery. After perforating the muscular coat, this artery passes downwards towards the anal canal along a line separating the left anterior from the left posterior quadrant, and ends in a meshwork in a Column of Morgagni at this point, the accompanying veins giving rise to the *left primary haemorrhoid*. This artery gives off two branches, an anterior and a posterior. The veins accompanying the former give rise to *the left anterior secondary haemorrhoid* and those accompanying the latter form *the left posterior secondary haemorrhoid*. The artery associated with the left anterior secondary haemorrhoid very rarely gives off a small branch which is distributed to the middle line anteriorly where an additional haemorrhoid is occasionally found.

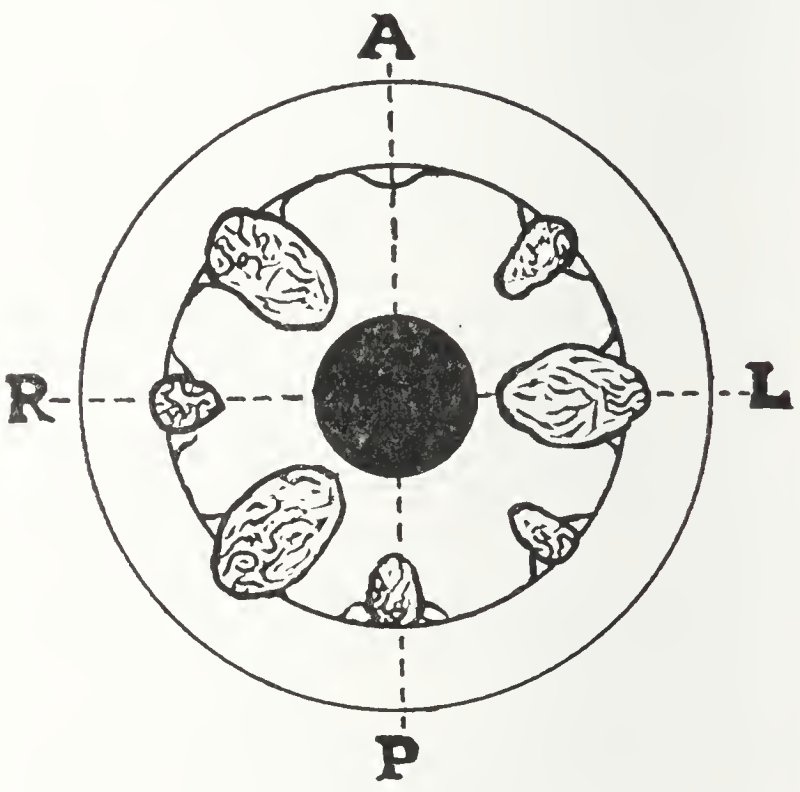

FIG. 2.—Diagram showing the secondary internal piles—right, posterior, left posterior and left anterior.

When pathological changes take place in the haemorrhoidal vessels, rendering them tortuous, dilated and thickened, those first affected are the larger or main branches. The smaller or secondary branches become involved later. Consequently the first vessels to be affected are those distributed (a) to the right anterior quadrant, (b) to the right posterior quadrant, and (c) to the left point in the anal circumference. Hence, haemorrhoids resulting from changes in the above mentioned vessels may be spoken of as **primary haemorrhoids.** Similarly, haemorrhoids developing in connection with the secondary branches may be considered to be **secondary haemorrhoids.**

In actual practice the presence only of

the *three primary haemorrhoids* is revealed in over seventy per cent of the cases examined, however the possible presence of the other four, and rarely five, secondary haemorrhoids should be borne in mind. The secondary haemorrhoids are, moreover, closely connected with the primary haemorrhoids from which they developed, so that in those instances in which all of the possible haemorrhoids are present, they are formed into three groups, viz:—

Group 1—The right anterior primary internal haemorrhoid.

Group 2—The right posterior primary haemorrhoid with two off-shoots or satellites, the right and the posterior secondary haemorrhoids.

Group 3—The left primary haemorrhoid with its two, or rarely, three off-shoots or satellites, namely, the left posterior, the left anterior and the anterior secondary haemorrhoids.

To complete the arterial supply of the rectum and anal canal, one should mention the middle and inferior haemorrhoidal arteries: the former a small branch of the anterior division of the internal iliac artery and supplying the middle of the rectum anastomosing with the superior haemorrhoidal tributaries; the latter arising from the internal pudental in Alcock's canal, and supplying the ischio-rectal fossa, the external sphincter muscle, the lower part of the anal canal and the peri-anal skin.

Venous Drainage of the Rectum and Anal Canal

The haemorrhoidal veins, superior, middle and inferior, drain the rectum, the anal canal, the external sphincter muscle, the ischio-rectal fossa and the peri-anal skin. The *superior haemorrhoidal vein*, the most important one, begins in the *internal haemorrhoidal venous plexus* (Fig. 3) in the submucosa in the vicinity of the pectinate line, collecting veins passing upwards in the submucosa of the Columns of Morgagni. They pierce the rectal wall about three inches from the anus and having reached the outside of the rectum, the several tributaries unite to form a single superior haemorrhoidal vein which accompanies the artery between two layers of the pelvic meso-rectum and the pelvic meso-colon, and crossing the left common iliac artery it becomes the inferior mesenteric vein terminating in the splenic vein. The latter with the superior mesenteric vein forms the portal vein. The *middle*

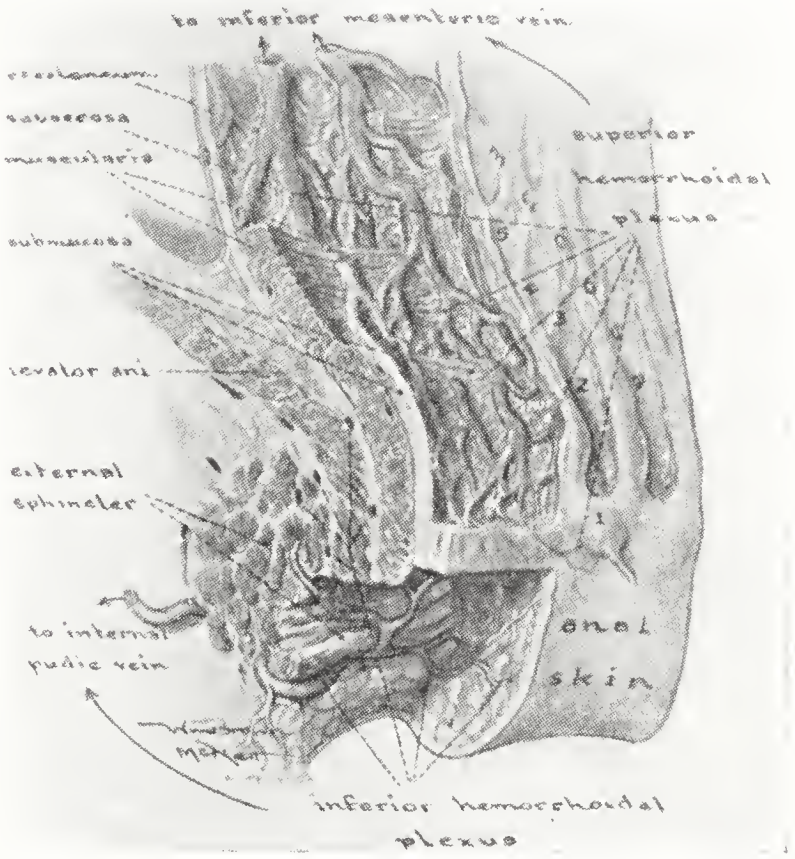

FIG. 3.—The venous drainage of the rectum and anal canal showing the derivation and structures.

haemorrhoidal vein arises in the submucosa of the rectal ampulla. They are usually small in size and pass outwards along the lateral ligaments of the rectum in company with the corresponding arteries to terminate in the internal iliac veins. The *inferior haemorrhoidal vein* is made up of tributaries which arise chiefly in the external sphincter, in the walls of the anal canal, and in the *external haemorrhoidal venous plexus* which lies immediately under the skin of the anal margin. This vein drains into the internal pubic vein and so to the internal iliac vein. Communicating veins exist in the anal canal between the external haemorrhoidal plexus (systemic venous system) and the internal haemorrhoidal plexus (portal system).

Etiology

Haemorrhoids occur most frequently during the fourth and fifth decades of life. It is said that seventy per cent of individuals who have reached the age of forty years suffer from haemorrhoids to some extent, but in only a small proportion are they sufficiently pronounced to require surgical treatment. Sedentary occupations seem to pre-dispose to their development, yet on the other hand heavy work involving great straining is a potent factor. The most important single factor in the production of haemorrhoids is habitual constipation. The irritation and straining associated with diarrhoea have the same effect, especially if this is associated with much weight loss. In these cases the support afforded by the ischio-rectal fat deposits to the long venous channels is greatly reduced. An increase in intra abdominal pressure, such as the presence of ascites, large pelvic neoplasms, the pregnant uterus, and chronic passive congestion with liver enlargement, will impede the venous return from the lower rectum.

Stages in the Development of Internal Haemorrhoids

Once haemorrhoids have developed, they become progressive and pass through three distinct stages, namely, (a) primary or first stage, (b) intermediate or second stage, (c) the final or third stage. It does not necessarily follow that in any given case all of the haemorrhoids present are in the same stage of development.

The primary stage. In this early stage the haemorrhoid is small and covered by healthy mucosa. The tumour is not palpable and only the lower pole of the swelling is seen on straining. A little red blood may be seen on the gloved finger after examination. The only symptom is *bleeding* and the blood is red. The condition is *painless*.

The intermediate stage. The haemorrhoids have become progressively elongated and more bulky due to exudation into the areolar tissue and increased dilatation of the component vessels. The tumour now is readily seen on visual examination after straining. They protrude at each evacuation and become spontaneously reduced as soon as the expulsive effort has ceased. On account of repeated protrusion, the mucosa becomes thickened and loses its original bright red colour. The dilated vessels being protected by the thickened mucosa, bleeding is less frequent and less copious. It is claimed by a few proctologists that the tumour "can be detected by the examining finger as a thickened longitudinal fold, especially if the finger is rotated." I am not in agreement with this statement. I do

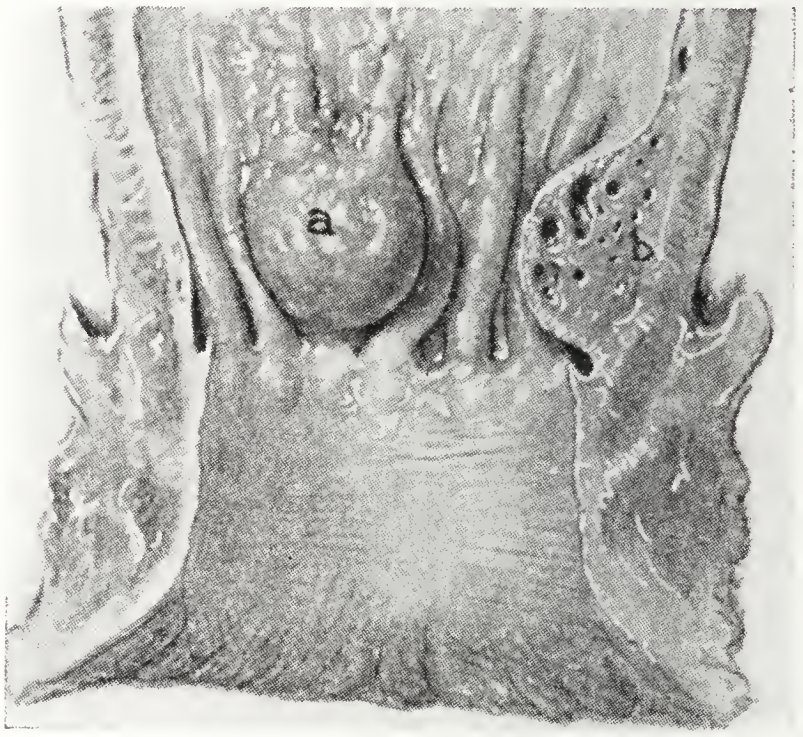

FIG. 4.—a. Internal haemorrhoid. b. Same on cross section.

not believe that one can detect a haemorrhoid by digital examination alone unless it is thrombosed. One might define the term "internal haemorrhoid" as a tumour situated in the anal canal covered by mucosa and containing several dilated veins, one or more arteries, interstitial tissue and lymphatics (Fig. 4).

The final stage. The protrusion is now pronounced, is continuous and does not spontaneously become reduced. It is necessary to reduce it digitally after each act of defaecation and recurrence of the protrusion takes place on slight provocation, such as passing of flatus, coughing, sneezing or standing for any length of time. As the mucosa from constant external exposure becomes

thickened and tough, bleeding is not so common and when it occurs, is due to trauma or to one or more haemorrhoids not having reached the final stage. As mentioned elsewhere, it is a common occurrence to find the three stages represented in one case.

Symptomatology

The earliest and most constant symptom of haemorrhoids is just what one should expect, viz.: *bleeding* with or immediately following evacuation. It is *painless*. It first is quite red, later, it becomes darker in colour. It is indeed amazing to find patients who will allow this progressive loss of blood to continue without seeking medical advice. On the advice of a misinformed friend, they seek relief through the liberal use of suppositories and even internal "medication." As the condition progresses, protrusion will be added to the above symptom. If this state of affairs is allowed to continue over a period of several months or years, a state of *anaemia* is almost certain to develop. In order to emphasize the importance of this last symptom I shall take the liberty of giving you the story of two cases seen during the past year; one, a farmer about fifty, was referred to me by his physician. He had had "bleeding piles" for two years but had only recently confided this information to his physician. My first impression, after looking at his face, was that he had a carcinoma of the rectum, so pale and yellowish was his colour. Fortunately however, he was suffering from internal haemorrhoids of the intermediate or second stage. His haemoglobin was 55 per cent, red blood count 3,200,000. The other case was that of a soldier of the last war who had been suffering from haemorrhoids since his discharge from the army some twenty years ago. His pallor was goose-flesh in appearance, not unlike a case of pernicious anaemia. He suffered from internal haemorrhoids of the third stage. Haemoglobin was 60 per cent, red blood count 2,600,000. On proctoscopic examination both cases were negative for malignancy. Operative treatment put both these men back on their feet.

In the case of the final stage of haemorrhoids, a third symptom is added, viz: *mucous discharge*. This is a troublesome symptom and causes undergarments to become adherent to the protruding mucosa so that dried and fresh blood will be seen on these. The recent blood, of course, is caused by tearing free the adherent garments. A further symptom is frequently added at this stage, viz: *symptomatic pruritus ani*, due to the constant irritation from mucus secreted and poured over the adjacent skin by haemorrhoids which are "not at home."

Treatment of Internal Haemorrhoids

Before considering the treatment of internal haemorrhoids, let me remind you that fifty per cent of patients who come to us complaining of "haemorrhoids" or "piles" have not haemorrhoids at all, but some other condition such as pruritus ani, ischio-rectal abscess, fistula-in-ano, anal fissure, anal papilloma, rectal carcinoma or a pilonidal cyst or sinus.

A *digital rectal examination* should precede any form of treatment contemplated. Why? (a) To ascertain the possibility of thrombosis, in which case the haemorrhoids will be *palpable*. (To my mind this is the only type of internal haemorrhoid which can be felt on digital examination alone.) (b) To rule out the possibility of a neoplasm. If a rectal neoplasm is the cause of haemorrhoids it must be situated at or below the point where the venous channels leave the rectum, otherwise it cannot impede the venous drainage. We have seen that the superior haemorrhoidal veins leave the rectum about three inches from the anal orifice. It is therefore clear that such a tumour, if present, should be felt by the examining finger. If haemorrhoids are present in a case of carcinoma above this level, they are caused by straining at stool and not by mechanical obstruction of the veins. However, if digital rectal examination is negative and there are reasons to suspect from the history (constipation, diarrhoea, loss of weight, anaemia) that the patient has a malignant growth

higher up, it is your duty to carry out a proctoscopic examination, or further, a barium enema.

The treatment of the *primary stage* is definitely non-operative. It consists of the injection of a sclerosing fluid under the mucosa of the haemorrhoid-bearing area. The solution is not injected into the lumen of the vein but in the submucosa adjacent to it; it is practically

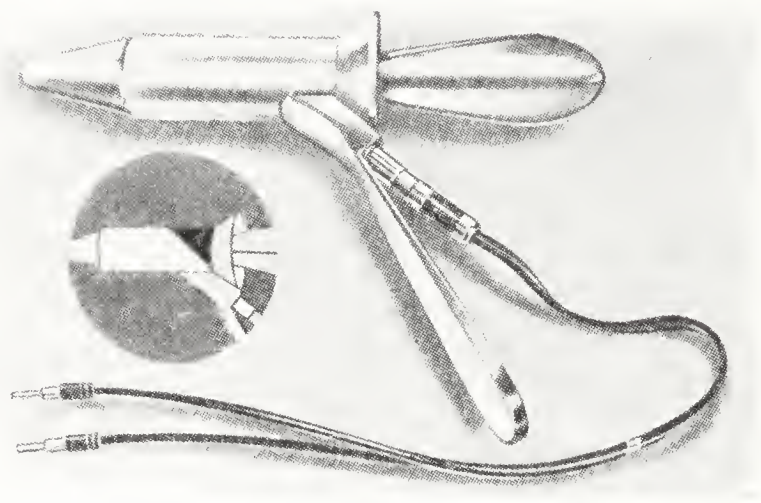

FIG. 5.—Illuminated proctoscope.

impossible to identify any one vein large enough to accomplish this and moreover, there would be danger of embolism. The only preparation desirable is a recent evacuation of the bowel. No cathartic is necessary. The *equipment required* consists of an anoscope with direct lighting by means of a battery or indirect current. My personal choice is a Brinkerhoff anoscope of medium size (another type is illustrated in Fig. 5). It has a tapering end and is provided with a slide which is partially withdrawn after the instrument has been fully inserted and the bulging mucosa is visualized as it drops into the slot of the illuminated lumen. The light is hooked into a small elbow soldered at the side of the proximal end. A needle four inches long and number twenty gauge is securely fastened to a five cc. syringe. I have obtained excellent results from the use of five per cent phenol in almond oil as the sclerosing fluid. About 1.5 cc. is used at each injection. The lubricated anoscope is fully inserted and the slide is partially withdrawn. The injection is made high up in the submucosa of the haemorrhoid (Fig. 6), and it is best to make sure you have not accidently punctured a large vein by first drawing back on the plunger. If there is blanching on injection, the needle is too superficial; if no swelling occurs, the needle has buttonholed the mucosa or is too deep. After completing the injection, the needle is left in position for about ten seconds to prevent leaking back through the puncture. In charting the treatment, it is convenient to use the term "9 o'clock, 3 o'clock, etc." The patient is permitted to carry on his usual occupation but is instructed to take things easy for the rest of the day. No other instructions are necessary. The treatment is given once weekly for four weeks, and a digital rectal examination should precede each injection. A thrombosed haemorrhoid is readily felt. The patient returns for examination one month after the last treatment but it is seldom necessary to give more than four injections. I have never seen any serious consequences following this type of treatment. Occasionally, ulceration occurs at the site of the injection, emphasizing the wisdom of digital examination prior to each treatment. Ulcera-

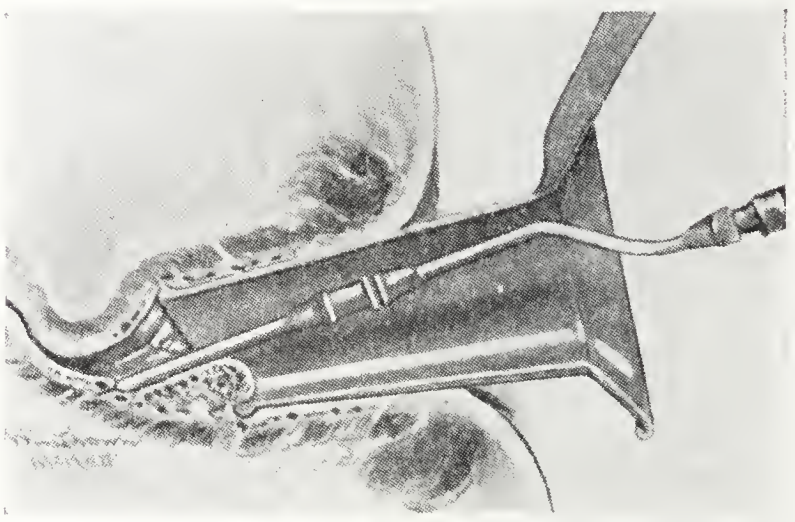

FIG. 6.—Lateral view of the needle in the upper portion of the haemorrhoid, preparatory to injection of the sclerosing solution.

tion means postponement of further treatment for obvious reasons.

The treatment of the *intermediate stage.* Approximately fifty per cent of these may be satisfactorily treated by injection. Unfortunately, however, internal haemorrhoids at this stage are frequently associated with external tags, a complication which necessitates operative treatment as the latter cannot be successfully treated in any other way. The operation practised by the writer is

that of excision and suture, a brief description of which will be given subsequently. Many surgeons still use the clamp and cautery method for internal haemorrhoids. Unfortunately however, external tags cannot be removed by this method because a burn on the skin is very painful and healing is unduly prolonged.

FIG. 7.—Prolapsed right anterior and right posterior piles of the third degree.

The treatment of the *final stage* is definitely operative as the internal haemorrhoids are always associated with external tags (Fig. 7).

Operative Treatment of Haemorrhoids

The primary purpose of operative treatment is the excision of haemorrhoidal tissue in addition to the skin tags at the anal orifice, should these be present. In cases where the latter are not present, it is wise to excise a small triangular portion of skin in association with the primary internal haemorrhoids. In the majority of instances, it is necessary to excise all three primary haemorrhoids with one or two secondary haemorrhoids adjacent to them. A diamond shaped incision is planned to include the skin tag and the associated redundant mucosa which contains the dilated veins. In Fig. 8 the lower picture shows the diamond-shaped excision in progress, the outer angle of the diamond being held by forceps, the haemorrhoid-bearing area being cut away by means of a scalpel over a crushing clamp which has been applied to the apex of the diamond on its anal aspect. The stump is now transfixed with plain catgut, the short end of the suture being tied above the clamp. The next suture is passed just behind the first and a loop is thrown snugly above the clamp. A third suture is passed over the clamp which is then released. The stump is then buried by another suture at the anal angle of the diamond. It is best not to include the sphincter fibres in this suture. Whether the sides of the incision are opposed by a subcutaneous suture or not, depends upon the danger of constriction of the anal orifice. This suture line secures all bleeding points which otherwise should be tied. After completion of the opera-

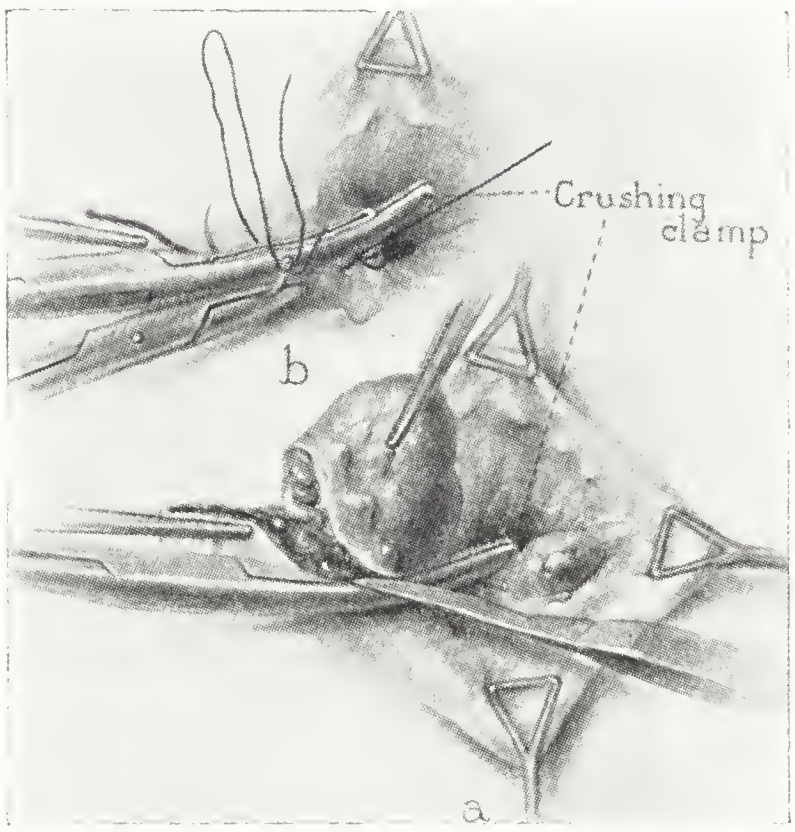

FIG. 8.—Buie's operation. a. Crushing clamp applied and excision of the haemorrhoid. b. Haemorrhoidal stump sutured.

tion the suture lines are viewed by means of an anoscope; additional bleeding points must be tied and a penrose drain inserted into the anal canal. Dressings are applied by means of a T-binder. The post-operative care is most important. The routine followed by the writer is as follows:—

1. Warm boracic compresses are applied to the sides of the anal orifice every four hours.

2. Morphia gr. ¼ per hypo, every four hours for twenty-four hours.

3. Catheterize every twelve hours if necessary.

4. Full diet as soon as tolerated.

5. Bathroom privileges, day after operation.

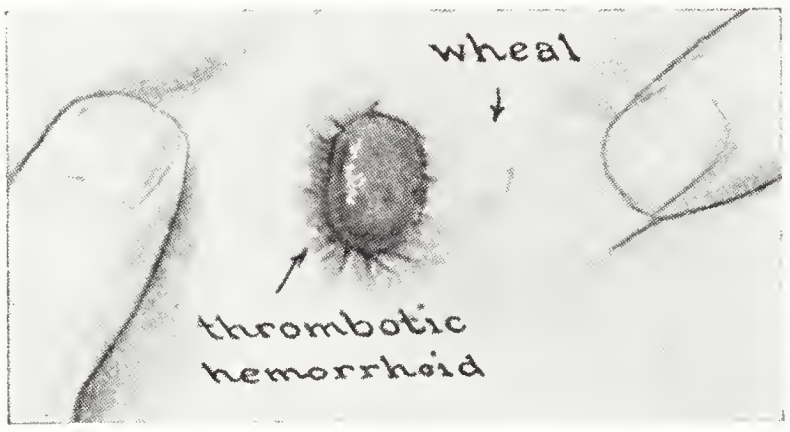

FIG. 9.—A small wheal has been raised just outside the thrombotic haemorrhoid. Through this the procaine solution is injected beneath the pile.

6. Cathartic, night of second day post-operative. Warm olive oil enema, four ounces, followed by a 1, 2, 4 enema (at least twenty-eight ounces) next morning. Then cathartics as required.

7. Anal irrigation with normal saline twice daily and after bowel movements, and continued for seven or eight days.

8. A digital rectal examination should be done on the sixth day and every day or two after that up to the tenth or twelfth day.

9. Patients are allowed to leave hospital at the end of a week.

Post-Operative Complications

Haemorrhage. If the operation has been properly performed, this is indeed a rare complication. Oozing from the wound surface is easily arrested by a firm dressing but frank haemorrhage cannot be so controlled. Bleeding is usually of the reactionary type, occurring a few hours after operation. Blood collects above the sphincter and is therefore hidden from view, and if the bleeding continues, the patient presents a typical picture of reactionary haemorrhage. A nurse walks into the ward and finds the patient blanched, if not unconscious or gasping for air. The early sign may be an involuntary bowel movement of bright red and clotted blood. Such a case should be rushed to the operating room and the bleeding point ligated. One cannot do this on a sagging mattress or under a poor light; it is not only cruel but a waste of time to attempt to stop it without an anaesthetic.

Narrowing of Anal Canal. This complication should not occur either if the lumen has been left adequate after operation and careful post-operative care is followed.

Thrombosed External Haemorrhoids

This condition is actually an extravasation into the perivenous spaces at the anal orifice. The onset is acute and painful. A cold stone or the excessive use of alcohol is usually blamed for its development. They are not necessarily associated with internal haemorrhoids. The treatment consists of an incision into the swelling under local infiltration of novocaine and removal of blood clots (Figs. 9 and 10).

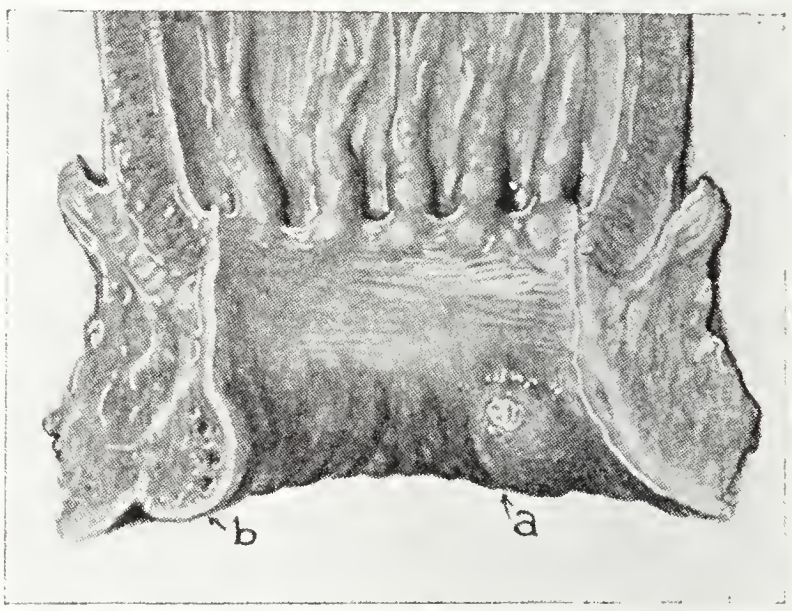

FIG. 10.—Coronal section of lower rectum and anal canal showing a. external varicose haemorrhoid; b. same on cross section.

Prolapsed, Thrombosed and Ulcerated Haemorrhoids

Prolapsed internal haemorrhoids are frequently subjected to irritation and trauma. Oedema of the protruded mucosa develops and renders replacement increasingly difficult. Pain causes spasm of external sphincter muscle and

further interference with venous and lymphatic drainage. Under such conditions, venous thrombosis is certain to follow and extends, via the communicating veins, to the external haemorrhoidal plexus with oedema of the peri-anal skin (Fig. 11). Attempts at reduction at this stage are not only futile but harmful. Arterial thrombosis which follows, results in moist gangrene of most of the protruding mucosa and the picture to the inexperienced eye is not unlike that of a neoplasm.

FIG. 11.—Prolapsed, thrombosed and ulcerated internal piles.

Treatment. Most of these cases are seen too late to attempt digital reduction. I am not in favour of wide cutting operations. For some time now I have successfully treated these cases palliatively. The patient is tired from loss of sleep, has not taken any food for two or three days, and neither has he had an evacuation of the bowels. He is put to bed with the foot elevated to assist drainage; is given no cathartic but a clearing enema; morphia gr. 1/4 every four hours for twenty-four hours; soft diet; cold saturated magnesium sulphate compresses are applied every four hours to reduce the swelling. It is amazing how the patient will admit improvement in a few hours. It is permissible, under local anaesthesia, to excise one or two extremely painful blobs of gangrenous mucosa but no more. Improvement is progressive and in six to twelve days the swelling has almost completely subsided so that he may leave the hospital and return for a radical haemorrhoidectomy in two or three weeks.

External Haemorrhoids

This is a very unappropriate term and justification for its use is seldom found clinically. Normally under the peri-anal skin lies the external haemorrhoidal plexus of veins. To my mind there are only two surgical conditions in which these veins are affected. The most frequent one is the so-called "thrombosed external haemorrhoid," mentioned elsewhere in this article, (Figs. 9 and 10) which is actually a peri-venous extravasation and leads to a small, painful swelling, bluish in colour, at the anal orifice. Its onset is acute and it does not necessarily occur in a pre-existing skin tag and is not usually associated with internal haemorrhoids. The other condition is thrombosis, extravasation and oedema occurring at the anal orifice and almost, if not completely, involving the whole circumference in cases of "thrombosed internal and external haemorrhoids" (Fig. 11). A condition which is frequently present at the anal orifice is excess skin which may be thrown into folds in the form of tags. They are simply redundant bits of skin and should not be termed external haemorrhoids. These skin tags are nearly always associated with long standing cases of internal haemorrhoids and are also seen in cases of thrombosed external haemorrhoids which have been allowed to subside without operation, leaving a firm fibrotic nodule, or nodules, at the peri-anal margin. They are occasionally associated with pruritus ani, either as a causative factor or secondary to the presence of pruritus.

Treatment. The treatment of skin tags and excess peri-anal skin is excision and as they are usually associated with internal haemorrhoids they are included in the excision. If they are associated with pruritus ani they should be excised as a preliminary step in the determination of the cause of the pruritus.

COMA

By EDWARD F. BROOKS, M.D., M.R.C.P. Lond.
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In this brief discussion of Coma, I propose to consider the more common conditions which may cause this state, and to outline a method of approach to the establishment of a diagnosis. I will discuss the question of diagnosis in the case of a patient whom you may be summoned to see, who shows the characteristics of this physical state we refer to as coma. One must first distinguish between sleep, stupor and coma. In true coma, the patient is unconscious and cannot be roused, the pupils are usually inactive, reflexes are absent and the patient is unable to swallow. Students in Medicine, who rely strictly on mechanized memory at the expense of intelligent reasoning, have reduced this problem in diagnosis to the simplicity of recalling the five vowels, a, e, i, o, u, and then, too often with difficulty, search their memory for the diagnosis which these letters should represent.

When one is presented with the problem of why a patient is in a state of coma, consideration of the possible causes should be made in logical order, and the student should acquaint himself with certain characteristics which will be of diagnostic value in different cases, according to the cause.

The chief causes to be considered in any case of Coma are:—

1. Injury

If the injury is the cause of a patient being in a state of coma, the history of course, will be of value.

Are there signs of trauma to the face or scalp?

Is there bleeding from the nose or ears?

Look for possible escape of spinal fluid from the canal of the ear. A blackish, mahogany colour to an intact ear drum suggests the presence of blood in the middle ear, as in fracture of the base of the skull.

If the patient has received a head injury which produced a transient concussion, to be followed by a lucid interval and now the coma in which you find him, the diagnosis of *Middle Meningeal Haemorrhage* is most probable. Unequal pupils, with the dilating pupil being on the side of the lesion, is an important observation in such a case.

Is the blood pressure rising?

Is the pulse becoming slower, the breathing stertorous?

These are signs of cerebral compression, and should be checked closely in all cases of coma.

Acute or sub-acute *Sub-dural Haematoma* may be the lesion present, or the patient may be showing signs of brain laceration, complicating a fractured skull. X-rays of skull, spinal puncture, careful check on pulse, blood-pressure, respirations, etc., should supplement a careful neurological examination in all cases of head injury.

2. Poisons

(a) External Poisons

Has the patient taken an overdose of drug?

The commonest causes in this group are:—

(i) *Alcohol*

An odour of alcohol on the patient's breath is not sufficient evidence to establish a diagnosis. It is common practice for a lay person to attempt to revive an unconscious patient by giving him brandy, and detecting the odour of such has led to a wrong diagnosis in many cases. Also one should remember that a person inebriated is particularly prone to accidents, and the comatose condition you find him in, may be the result of a head injury, complicated by intra-cranial haemorrhage.

All alcoholic cases who have any signs of head injury should be kept under close observation for a period of 24 hours. In true alcoholic coma, the pupils are usually dilated, the tendon reflexes are normal, and there is a sub-normal temperature.

(ii) *Opium Compounds*

Morphine is the more common drug of this group as a causative agent in the production of coma. In a case of Morphine poisoning, the temperature is normal, the skin is moist, the pupils small, and the respirations slow. The reflexes are usually normal.

(iii) *Nembutal and Veronal*

Other narcotics are becoming more popular in their use for suicidal purposes, e.g. Nembutal and Veronal. In veronal poisoning, the signs are similar to morphine poisoning, but the pupils are dilated.

(iv) *Carbolic Acid*

Carbolic Acid in large doses may cause coma.

(b) Internal Poisons

(i) *Diabetic Coma*

Usually in such cases, the patient has been diagnosed as being a diabetic, but even to-day cases may come to the coma stage without diagnosis. If possible, a blood sugar will be estimated in all cases of coma, and of course a urine examination for sugar and acetone is routine.

Can acetone odour be detected on the patient's breath?

(ii) *Uraemic Coma*

This rarely comes on suddenly, but usually as an end result, and we are prone to think too much of this diagnosis in such cases which suddenly go into coma.

(iii) *Cholaemia*

Cholæmia due to hepatic failure as may be seen in cases of Cirrhosis of the Liver, Secondary and Primary Carcinoma of the Liver. In most cases jaundice is present, but cholaemia may be present without jaundice.

(iv) *Hypoglycaemia*

In a known diabetic patient who is taking insulin, this diagnosis is readily considered, and usually does not present real difficulties. Examination of the urine, and if possible a blood sugar estimation is carried out, and the administration of glucose by mouth or intravenously, quickly establishes the diagnosis by restoring the patient to consciousness. One should remember that hypoglycaemia may occur in other than diabetic patients who are taking insulin. Cases of Simmond's Disease, and adenomata of the pancreas, are subject to periods of unconsciousness because hypoglycaemia is a feature in these cases, and coma may be a presenting symptom before a diagnosis of either of these conditions has been made.

3. Cerebral Apoplexy

Cerebral Thrombosis, Embolus, Haemorrhage, Angiospasm, or Subarachnoid Haemorrhage.

When a person past middle age suddenly becomes unconscious, the diagnosis of Cerebral Apoplexy should be our first consideration. Except in cases of pontine haemorrhage, or subarachnoid haemorrhage, signs of unilateral paralysis are usually present. Inequality of the pupils is common, the pupil being dilated on the side of the haemorrhage. Look for facial asymmetry. Is there a positive Babinski response on one side? Bilateral dorsiflexion response to the plantar reflex is common in any case of coma. Elevation of temperature and stertorous breathing are suggestive findings in cases of Cerebral Haemorrhage.

4. Post-Epileptic Coma

In these cases the previous history of the patient is important.

Has he had attacks of Epilepsy, or was the present comatose state preceded by a convulsion?

Has he bitten his tongue?

Has he passed his urine?

There is nothing characteristic about this coma stage, except that it is usually of a comparatively short period, often to be followed by sound but normal sleep.

The above are the more common causes of coma, but mention should be made of

other conditions which may result in the unconscious state.

5. Cerebral Neoplasm

When haemorrhage occurs in a cerebral tumour, coma may result, and is of sudden origin. The presence of a neoplasm may not have been suspected until this sudden event took place.

6. Inflammations

Coma in such instances usually occurs as a terminal event in cases of Encephalitis or Meningitis.

7. Sunstroke or Heatstroke

Characteristic of these cases is the high fever of 105 to 107°.

8. Malaria

In tropical countries the onset of coma might suggest the diagnosis of Malaria, etc.

I have briefly discussed the more common conditions which may be the cause of a patient being found in a state of coma. The physician should have an organized procedure to follow, to assist him in arriving at a diagnosis when such problems are presented to him.

Procedure

1. *History*

As in all medical and surgical problems, a careful history becomes primarily important. In a case of coma, you will seek all information available from the relatives or eye-witnesses. You may obtain information as to possible injuries, diabetes, epilepsy, the taking of insulin, etc.

2. *Evidence of Overdose, etc.*

Take a look around to see if you can find any evidence that the patient may have taken an overdose of medicine, e.g. medicine bottles. Do not disregard the possibility that a medicine bottle regularly labelled, with doctor's instructions thereon, may have been used by the person to carry wood alcohol. Also it may be advised here, that in the case of a patient who has morbid ideas, do not prescribe sedative drugs in amounts more than necessary for his immediate needs.

3. *Injury, Cerebral Apoplexy, etc.*

Examine carefully the scalp for evidence of injury. Examine the ear canals and drums. Are the pupils equal, constricted or dilated? Do they react to light? Do not dilate the pupils with mydriatics, as you may mask the presence of a pathological dilatation, which is a valuable sign in such conditions as Middle Meningeal Haemorrhage, etc. Look for asymmetry of the face. Examine the patient's breath for odours of acetone, alcohol, etc. Do not too readily diagnose alcoholic coma on detection of alcohol on the breath. Brandy may have been given as a stimulant, or even if intoxicated, he may be in coma because of injury, diabetis, etc. Examine for paralysis. Check the reflexes carefully. Positive Babinski responses are common in coma of any cause.

4. *T. P. R. Records*

Careful records of temperature, pulse and respiration will be of assistance.

5. *Urine*

Pass a catheter and examine the urine for sugar, acetone and albumin. Remember that any case of coma may show albumin, and cerebral lesions may show glycosuria. Glycosuria without acetonuria is suggestive of a cerberal lesion, or may be the finding in a coma of insulin overdose, particularly if the patient had not voided for some time.

6. *Stomach Lavage*

In any case in which poisoning is a possibility, pass a stomach tube and wash out the stomach.

7. *Spinal Fluid*

A spinal puncture may be necessary to establish a diagnosis and as an aid to treatment.

8. *Eyes*

Examine the fundi of the eyes in all cases of coma, and evidence may be found to assist in the diagnosis of such conditions as diabetes, uraemia, and cerebral vascular disease.

I have not attempted to discuss in detail the various conditions which may result in coma. This short paper has been written to assist the student in organizing a proper approach to this problem of diagnosis. I have enumerated the more important clinical conditions in which coma occurs as a presenting symptom, and not from the standpoint where coma occurs as a terminal event. In the end stages of many chronic conditions such as carcinoma, tuberculosis, etc., coma may be present. The student should recall too, that a semi-comatose or a comatose state may occur in a patient showing signs of cardiac failure. Cerebral anoxia would be present in such cases, and is the explanation of reduced consciousness in these patients. After the student has familiarized himself with the above resumé on this subject, he should then review each condition mentioned, in more detail, in order that he will have the complete knowledge necessary to a diagnosis in each case.

Miscellany Rules of Health and Long Life

(1) Chronical Diseases last long, wear out the Constitution leisurely, and are accompanied with a slow Pulse; whereas acute ones soon terminate either in Death or Recovery, and are joined with a quick Pulse.

(2) The Scurvy is the Root of most chronical Diseases of the British Nation; and is a necessary Consequence of their Way of living almost wholly on animal Food, and drinking so much strong Liquors.

(3) Soft, thin, small, short, fair Hair, slender Muscles and Bones; soft Flesh; a white, fair, blanch'd or ashen-coloured Complexion; a fair, corpulent, flegmatick, cold Constitution; chilliness, especially in the Feet; a Readiness to catch Colds; and being subject to immoderate Evacuations of any kind, are certain signs of loose, flobby, or relaxed Nerves.

(4) Women of weak Nerves are very subject to Miscarriages. Their danger is increased by high Living, and indiscreet bleeding. The only Remedy for them, is drinking Bristol Water and Red wine, with a low and light Diet, going Abroad to get Air, and using Astringent Plaisters, and other proper medicines to corroborate their Bowels.

(5) The Weak and Sickly (as their Concoctive Powers fail in Winter, and recover in Summer) should carefully proportion the Quantity and Quality of their Food, to the Strength of them in the several Seasons.

(6) The fewer Cloaths one uses, the hardier he will be. Flannell and great Loads of Cloaths by Day and Night, relax the Fibres, and promote only Sweating, instead of the natural and beneficial Perspiration.

(7) The Weak, Sedentary and Studious, should frequently shave their Head and Face, wash and scrape their Feet, and pare the Nails of their Toes.

(8) People that read and write much, ought to do them standing, or in as erect a Posture as they can. And those who can go about any Part of their Studies walking, should do it.

(9) The Aged should (1) carefully guard against all the Injuries of the Weather; and (2) lessen the Quantity, and lower the Quality of their Food gradually, as they grow older; even before a manifest Decay of Appetite force them to it.

(10) As chronical Diseases are not brought on all at once; so they cannot be quickly removed. A gradual Corruption, must be gradually remedied, 'Tis contrary to the Nature of chronical Diseases to be quickly cured.

An Essay of Health and Long Life
By George Cheyne, M.D., F.R.S.
London—1725
Printed at the Golden Ball over against the Royal Exchange in Cornhill.

A PROBLEM IN DIAGNOSIS

By G. A. HENDRY, M.D.

Case History

Miss E. H. Age—14 years. Admitted to Ward "F" T.G.H.

This young girl was raised on a farm. She was in relatively good health until six weeks before admission to the hospital. The onset of her illness was characterized by burning on micturition associated with hesitancy. Of late, these symptoms had become increasingly distressing. Shortly after the development of dysuria, an indefinite resistance of the lower abdominal muscles was noted—later a definite firm swelling developed in this area. For approximately four weeks the patient had experienced recurrent attacks of mild lower abdominal pain. She had never menstruated.

According to her mother the patient had not been well since an attack of acute appendicitis one year previously. At that time she was taken to a nearby hospital where the appendix was removed. The immediate post operative recovery was excellent, but the patient never completely regained her appetite, tired readily, and lost ten pounds in weight during the year.

Physical Examination

General—Revealed a fairly well nourished young girl, who was not acutely ill. She cooperated well during examination.

Chest—Clear.

Breasts—Firm and well developed.

Heart—Normal—Blood pressure 120/76.

Abdomen—Of normal contour. There was a well healed appendicectomy scar in the right lower quadrant. A mass in the lower abdomen could be felt, and appeared to be arising from the pelvis in the midline to a point halfway between the symphysis pubis and the umbilicus. This mass gave the impression of a four months' pregnancy, though it was restricted in mobility, firm in consistency and regular in outline. It was not tender.

Pelvic—There was a normal distribution of pubic hair. The external genitalia were well developed. The hymen was not ruptured and no examination was attempted.

Rectal—Unsatisfactory—no definite information obtained.

The remainder of the physical examination was essentially negative.

Laboratory Findings

Urinalysis (catheter specimen)—negative.

Haemoglobin—75%.

White Blood Count—10,000.

Sedimentation Time—20 minutes (Friedlander Modification).

Temperature (admission)—100 degrees.

Differential Diagnosis

1—Malignant Ovarian Neoplasm.

2—Congenital Atresia with Resultant Haematometra.

3—Myomata Uteri—(rare).

4—Pregnancy—(unlikely).

Course and Operative Treatment

In view of the admission temperature and rapid sedimentation rate, an exploratory laparotomy was deferred for five days. During this interval the temperature returned to normal, but the sedimentation rate remained rapid. An Ascheim-Zondek pregnancy test was reported negative.

At operation the patient was first examined in the lithotomy position. The introitus was normal. There was no vaginal or cervical atresia. The tumor, apparently involving the uterus was the size of a four months' pregnancy, irregular in outline, firm in consistency, and limited in mobility. A uterine sound could be passed into the cavity of the uterus for a distance of approximately one and one-half inches.

The abdomen was opened through a low right paramedian incision. The superficial fat was found to be oedematous. The fascia, rectus muscle and peritoneum were invaded and greatly thickened by a chronic inflammatory process. The entire omentum and the mesenteries of the transverse and sigmoid colons were similarly affected. Several firm glands were palpable at the bases of these mesenteries. The caecum was not involved and there were no adhesions in the vicinity. There was a dense infiltration of the extra-peritoneal connective tissue extending from the inguinal ring on one side to the corresponding area on the other, and involving the anterior abdominal wall. The bladder could not be identified. This thickened, yet friable tissue did not have the appearance of a malignant growth. The cellular infiltration in the midline was so dense that it formed a mass which bulged into the peritoneal cavity above the uterus. Here it was adherent both to the uterine fundus and to the anterior abdominal wall, and accounted for the tumor felt on bimanual examination. The uterus itself was small and upright and both ovaries were healthy in appearance. It was impossible to excise this tumor. A section of the omentum was removed for examination and the abdomen was closed, without drainage.

Pathological Report

Specimen—Omentum.

Diagnosis—Actinomycosis.

"Microscopically, sections of tissue show it to be mostly of a type of chronic granulation tissue which is heavily overrun by numerous lymphocytes and plasma cells. Along one edge there are two typical colonies of actinomycosis having radiating eosinophilic club shaped forms."

Progress

For approximately two weeks the patient ran a septic temperature, with a maximum elevation of 104 degrees. Purulent drainage developed at the site of the retention sutures and cultures of the pus showed numerous typical colonies of actinomycosis. During her stay in the hospital she received a course of high voltage X-ray treatments and was given heavy doses of potassium iodide per os. Despite treatment the patient's general health failed considerably. After 6 weeks she was sent home by ambulance. The sinuses at the site of the retention sutures remained patulous, drainage was profuse and frequent dressings were necessary. The midline incision did not break down.

After four weeks at home she returned to the hospital for a second course of high voltage X-ray treatments. On admission, she was pale and emaciated and further abdominal sinuses had developed. Following treatment she returned to her home where her death soon occurred.

Discussion

The causative agent of this infection in human beings is the "actinomycosis bovis." The organism is transmitted from diseased cattle.

Thus the sequence of events in this case may readily be traced. This young girl was raised on a farm. Undoubtedly, she harboured the organism in her bowel at the time of the appendicectomy 12 months prior to her present illness. Such an operation would permit its escape into the general peritoneal cavity or abdominal wall.

The disease usually spreads by direct extension, rarely by way of the lymphatics. In the tissues the organism causes necrosis of cells which is accompanied by a great accumulation of lymphocytes and a later stage, is the formation of a thick wall of granulation

tissue. The central area is made up of pus containing the typical branching organisms. In this pus the characteristic "sulphur granules" are evident. Ordinarily the extension is slow but ultimately discharging sinuses develop.

The prognosis in extensive abdominal actinomycosis is extremely poor. Radical surgery is impossible and the results obtained by high voltage X-ray therapy are not encouraging. The disease progresses slowly, but relentlessly and death from general debility is the ultimate result.

The diagnosis of actinomycosis is always uncertain unless discharging sinuses are present. In this case the firm dense area of cellulitis overlying the bladder and adherent to the uterus gave the impression of being a malignant intraperitoneal tumor of uterine or ovarian origin.

Applications will be received up to April 1, 1940, for the following positions on the editorial board of the University of Toronto Medical Journal for the year 1940-41.

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ROENTGENOLOGIC EXAMINATION OF THE UPPER GASTRO-INTESTINAL TRACT

By S. M. HUDECKI, '40

With the discovery and subsequent development of the roentgen-ray in the last four decades, diagnostic accuracy in gastro-intestinal diseases has markedly improved. Much of the improvement has been due to refinements in the technique of examination.

In this paper a routine barium-meal examination will be discussed, as well as the more important diseases of the upper gastro-intestinal tract which lend themselves especially to radiological study.

The examination is both fluoroscopic and radiographic. An adequate series of radiographs confirms fluoroscopic findings, discovers undetected organic lesions and provides a permanent record.

There are certain time intervals after the ingestion of a barium meal which are radiographically important, affording the best opportunity for investigating successive portions of the alimentary tract.

(1) *Immediately After.* The oesophagus, stomach and duodenum should be studied at first after one mouthful of the medium to demonstrate the mucosal pattern. Immediately following this, the filled viscus should be examined.

(2) *Six Hours After.* This stage shows any pathological stasis in the stomach. Considerable six-hour gastric residue is usually of pathological importance. At this stage the terminal ileum, appendix, caecum and right colon may be studied.

(3) *Twenty-Four Hours After.* The appendix may be better demonstrated at this time than at six hours, and the colon to a degree varying with the distribution in it. Particularly the function of the colon is investigated.

(4) *Forty-Eight Hours After* and at succeeding daily intervals depending on the rate of colonic emptying.

These time intervals form the basis of a routine technique and are subject to variations to meet special cases.

A mixture of barium sulphate in water, flavoured with malted milk is used. The consistency of this preparation may be varied to suit special requirements. To examine the stomach as well as the oesophagus, two tablespoonsfuls of barium sulphate in ten ounces of water may be used. This can be made thicker if on preliminary examination it is found necessary.

The Technique of Investigation

Pharynx and Oesophagus

In a routine examination, the patient is placed in the erect position in the screening stand. The chest is first screened to exclude gross abnormality, then the patient is turned to the right oblique position. This brings the whole of the oesophagus into clear view as it descends in the posterior mediastinal space. The patient is instructed to swallow a mouthful of barium, the course of which is followed down the oesophagus fluoroscopically.

Following the screen examination, at least two radiograms of the whole extent of the oesophagus are taken, each immediately after swallowing a mouthful of barium meal. The exposure is as brief as possible to avoid transmitted cardiac movements.

To demonstrate the upper and lower limit of a lesion or to show the site of a pharyngeal pouch, the patient is examined in the horizontal position. Where no oesophageal lesion is suspected or observed under the fluroscope, radiographs are not necessary and the observer may proceed with the examination of the stomach.

Stomach

At this stage the patient having swallowed a mouthful or two of barium, is examined in the upright position. The barium is noted in the most dependent part of the stomach. With his right gloved hand the examiner pushes the barium gently into each part of the stomach in an attempt to "whitewash" the mucosa. This manipulation brings into relief the mucosal pattern of the stomach which cannot be demonstrated with the stomach more completely filled (Figs. 12 and 13). Any abnormality or suspected lesion calls for a radiographic confirmation. Lesions on the anterior wall or in the fundal region are best demonstrated with the patient in the prone or supine position.

The examination is continued by asking the patient to drink about ten ounces of the barium suspension, a quantity which almost completely fills the stomach. The capacity of the stomach varies in individual cases. With the stomach filled the following points are noted: *position, tone, peristaltic activity, flexibility* of the wall and presence of any *persistent irregularity* of the contours. This requires close observation only and no manipulation. The patient is then rotated into the right and left oblique positions, to bring into a profile view, the anterior and posterior wall of the stomach, noting especially any projections from the normal contour.

Attention is then directed to the pyloric region and the duodenum. When the stomach is filled with barium, succeeding peristaltic waves are seen to sweep towards the pylorus. The first few waves carry no barium, but then a small quantity is shot upward to the duodenal bulb. Two or three such waves are required to distend the bulb completely. The bulb retains its contents for a few seconds at most and then by a contractile wave, passes the bolus on into the villous portion of the duodenum.

The *bulbus duodeni* or the duodenal cap is an important radiological entity as it is the seat of duodenal ulceration. It has a "cocked-hat" or "acorn" appearance with a regularly curved right and left borders and the concave base. The latter is also regular in its curve, the concavity of the base being due to the bulge of the pyloric sphincter muscle and is increased in hypertrophy of the sphincter. The pyloric canal should join the base of the cap at its middle. The outline at the apex is surmounted by a feathery shadow caused by valvulae conniventes, beginning at this point. The cap and this variable amount of feathery duodenum i.e. duodenum supplied with valvulae conniventes, constitute the anatomical first part of the duodenum (Fig. 10).

To properly visualize this region, firm palpation with the gloved hand will push some of the barium into the duodenum. The palpation should be a massaging movement upward and to the patient's right and may be most effectively carried out with help of a peristaltic wave.

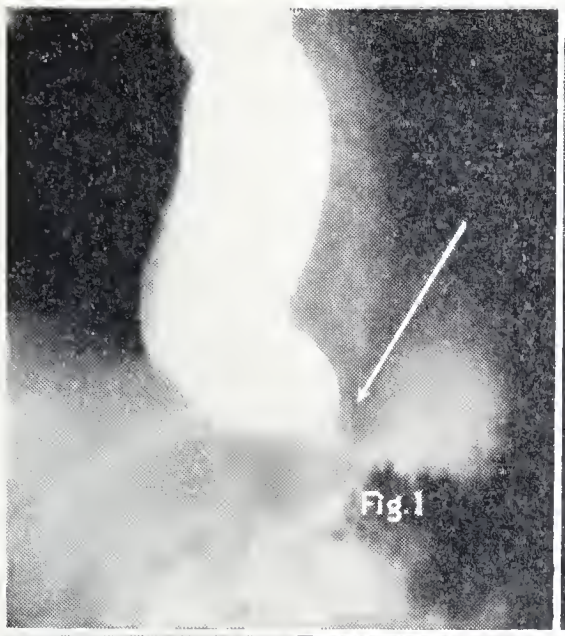
During this manoeuvre, the patient again must be rotated either to the left or right oblique position to separate the duodenal cap from the shadow of a superimposed pyloric antrum or second part of the duodenum. The latter condition is likely to exist with a hypotonic stomach.

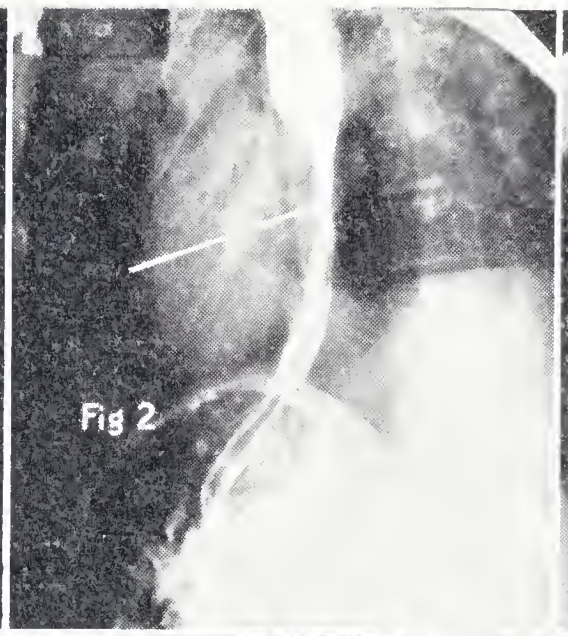
During fluoroscopy, the relationship of any *tender pressure points* to gastric and duodenal shadow should be noted and also the relationship to any *tumour mass* palpable clinically.

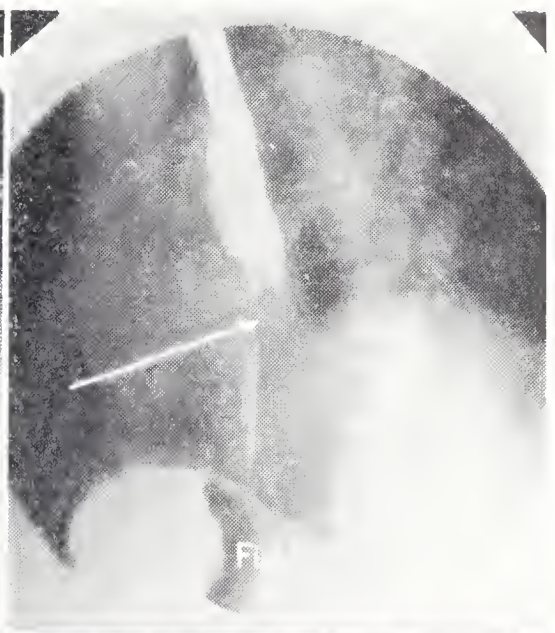
In the erect position, especially if the stomach is not completely filled with barium, an early filling defect may be readily missed in the fundal and upper cardiac region. This area is difficult to explore by palpation and manipulation of barium because of the costal margin. The patient is instructed to swallow six more ounces of barium and is examined in the supine position. The fundus then becomes dependent, well filled with barium and may be adequately examined.

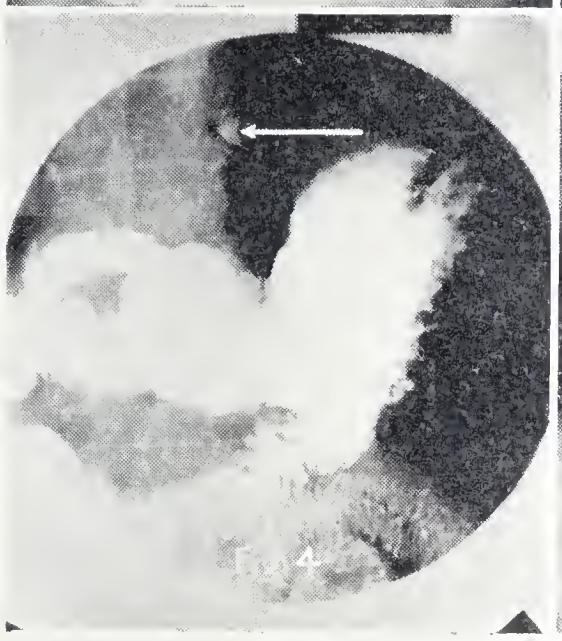
Radioscopic Examination

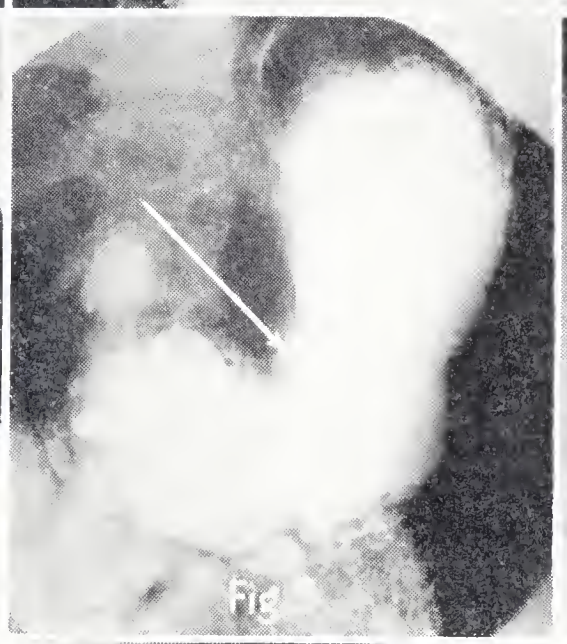
After the fluoroscopic examination, there remains the taking of a series of radiograms. These detect fine changes and confirm the organic nature of an abnormal appearance by its constancy. This constancy must be demonstrated by a

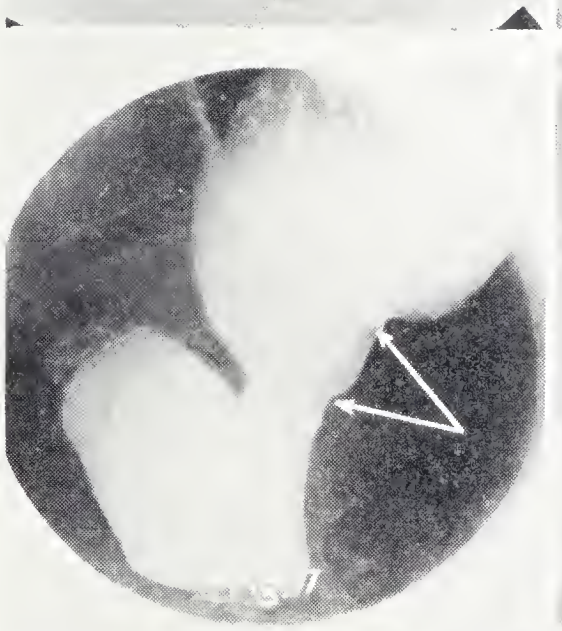


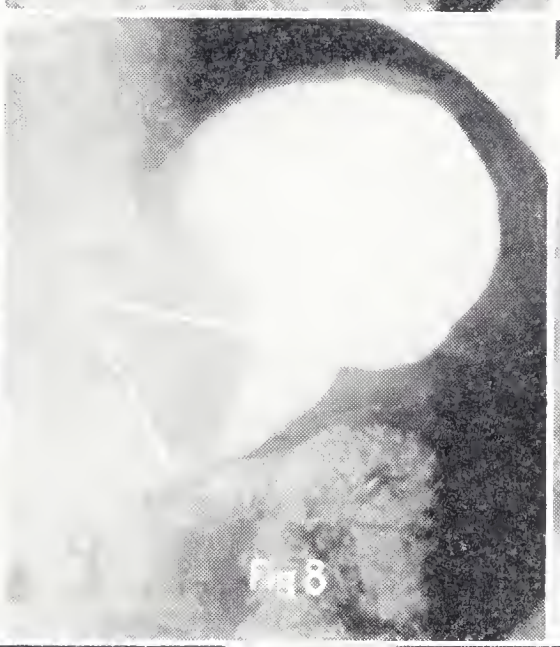


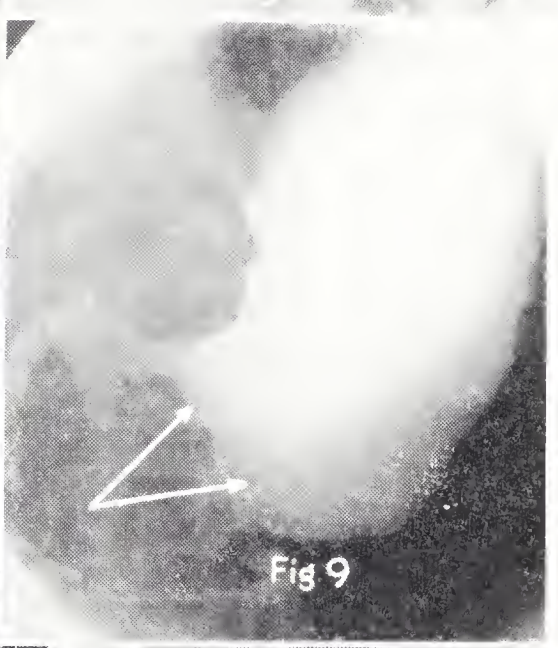












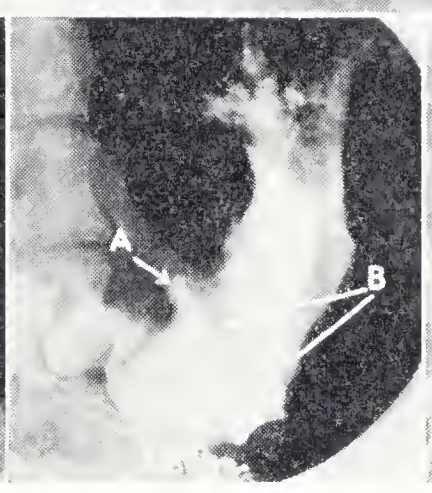


Fig. 1 Cardiospasm of Oesophagus; **2** Benign Stricture of Oesophagus; **3** Carcinoma, Lower Third of Oesophagus; **4** Ulcer at Lower End of Oesophagus; **5** Tripologram of Stomach (3 exposures on one film) showing the constant deformity of a Gastric Ulcer; **6** Duodenal Ulcer; **7** Early Carcinoma of Greater Curvature of Stomach, proven at operation; **8** Scirrhous Carcinoma of Stomach; **9** Advanced Adenocarcinoma of Pyloric End of Stomach; **10** Normal Stomach and Duodenum; **11** Gastric Ulcer on Lesser Curvature at "A", plus Extensive Carcinoma in Stomach Wall at "B"; **12** Apparently Normal Stomach (see Fig. 13); **13** (same case as Fig. 12), Mucosal relief pattern shows Polypoid Growth on Greater Curvature at "A". Cluster of three small Benign Polyps found at operation.

Films are the courtesy of Dr. Charles E. Vaughan, Assistant Radiologist, Hamilton General Hospital, and Radiologist, McGregor Clinic, Hamilton, Ontario.

series of radiograms or alternatively by making three rapid exposures on one film (Fig. 5). The latter method is the less satisfactory of the two.

The films are taken with the patient in a prone position and supported by pillows under the chest and hips so that the abdominal wall just clears the table. Under fluoroscopic examination the position of the patient is altered to bring into view the duodenum and stomach. Several radiographs are then taken in rapid succession. The exposure time is reduced to a minimum and the patient is instructed to stop breathing during the procedure. Taking the radiogram completes the first part of the examination, the patient is then asked to return for the six-hour examination.

X-Ray Appearances of Gastrointestinal Disease

Cardiospasm (syn. achalasia of the cardia)

Pathology—In a well developed case.

(1) Oesophagus is markedly dilated in its lower part. The dilatation gradually diminishes upwards, being almost non-existent in the cervical portion. The degree of dilatation is characteristic of this condition. No other obstructive lesion will produce this degree of dilatation.

(2) The oesophagus becomes lengthened, assuming an S shaped appearance, the lower convexity appearing to the left.

(3) The cardiac sphincter is in a state of tight muscular collapse. There is no actual hypertrophy of the sphincter. The location of the obstruction is not the cardiac end but is where the oesophagus enters the groove in the liver.

Radiological features (Fig. 1)

As the barium meal is swallowed it gradually forms a pool at the level of the diaphragm. The lower part of the dilated oesophagus is typically blunt conical, tapering smoothly to a point, the sphincter itself. The smooth regularity of the cone is a diagnostic point. The dilatation extends beyond the spinal column on the right side turning sharply to the left at its lower extremity. The obstruction is never absolute; when the oesophagus becomes full, some of the barium escapes into the stomach. It seems as though a certain "head" is necessary to relax the sphincter sufficiently to bring the barium column down.

Benign Stricture of the Oesophagus

A history of having swallowed corrosives, or a foreign body which become impacted or of oesophageal ulcers always suggests this condition.

Morbid Anatomy

Depending on whether the exciting factor involves a localized region or a more extensive one, the resulting stricture is either (1) annular or (2) tubular.

Radiological Features

Annular—The proximal portion of the oesophagus is dilated, terminating conically below into the stricture. There is very little irregularity above the stenosed area, and there are no spiky-irregularities to the stricture itself as in carcinoma.

Tubular Type (Fig. 2). A considerable portion of the oesophagus is involved. The narrowing varies from point to point. There is not as much dilatation because areas above the actual narrowing are involved to a lesser degree by the caustic. Any marked deviation of the oesophageal lumen indicates that peri-oesophageal tissue was involved by the original process. Again it lacks irregularity of neoplastic growth.

To differentiate it from the scirrhous type of carcinoma, a history of previous trauma may be the deciding factor.

Oesophageal Diverticula

Two types are described.

(1) *Pulsion diverticulum* or a more descriptive term *deep pharyngeal*

pressure diverticulum is the more common. It is a herniation of the mucous membrane through the transverse and oblique fibres of the inferior constrictor muscle of the pharynx, at a level one centimetre above the upper end of the oesophagus on the posterior surface in the midline.

X-ray Appearance

When observed in the oblique positions, the barium is seen to pass into a rounded *flask shaped pouch* and then spills over anteriorly down into the oesophagus. Characteristically the barium filled pouch shows a horizontal fluid level.

(2) *Traction Diverticulum* is very rare, resulting from traction of extrinsic scar tissue on the anterior wall of the oesophagus. It is best visualized in the oblique positions and fills best with the patient lying down prone.

Carcinoma of the Oesophagus

Morbid Anatomy (Fig. 3)

At first limited to the mucosa, the growth tends to become annular in distribution, causing a stenosis. Small excrescences and ulceration combine to produce a narrow tortuous channel.

Dilatation above the constriction is slight or absent because dilatation takes time.

Most of the growths occur in the middle third, where the left bronchus crosses. This number is about equalled by those of the upper and lower third combined.

Radiographic Features

The slight dilatation tapers abruptly and irregularly into the stenosed passage. This irregularity may be in the nature of a filling defect if the upper part of the growth is of the "cauliflower" type. The narrowing may extend over 2-3 inches requiring examination in the horizontal position to demonstrate its extent.

The characteristic feature of carcinomatous growth are its *irregularity* and *constancy of the irregularity*. This constancy is tested by the superimposition of successive films.

Syphilis of the Oesophagus

Cannot be differentiated radiologically from scirrhous carcinoma.

Oesophageal Varices

Oesophageal varices present a characteristic appearance in a radiogram. Using a fairly liquid medium with the patient lying down, the mucosal folds over the varicose veins are seen outlined with barium.

Oesophageal Ulcers

Ulceration of the lower 2 to 3 inches of the oesophague is found. The usual symptoms of gastric ulcer, relieved by medical treatment, or dysphagia alone may suggest a radiographic examination.

X-ray Findings (Fig. 4)

A small fleck of barium may remain constantly in the ulcer crater. An area of spasm or irritability at the level of the involved wall may produce slight delay to barium flow.

Gastric Ulcer

The barium meal is the only accurate method of investigation.

Pathological Features of a Chronic Gastric Ulcer

(1) Usually single.
(2) Position—on the lesser curvature including the anterior and posterior wall close to the lesser curvature, in 82% (Hurst & Stewart).
(3) Size. The Mayo clinic reports that 95% of ulcers are less than 2.5 cms. in diameter. This figure is not generally accepted.
(4). Description. In the *active stage* the appearance is that of an excavation in an oedematous mucosa with thickened overhanging edges. In the *healing stage*, the thickening and oedema of the edges disappears, and the ulcer assumes a conical shape.

Radiological Features

These are divided into two classes, direct and indirect. The direct sign is pathognomonic.

The Direct Signs of Gastric Ulcer

This consists in demonstrating the ulcer crater, the "niche" in the mucosal pattern radiogram or a protrusion of the barium shadow from the lesser curvature when the stomach is filled with barium and examined in profile.

Appearances of the crater in the mucosal pattern radiogram

With an ulcer on the posterior wall and the radiogram taken with the patient supine, there will be visible:

(1) A central fleck of barium which is constant in position and marks the barium-filled crater.
(2) A zone around this which is devoid of barium, representing the oedematous mucosal lip of the crater.
(3) A corona of mucosal rugae, produced as a result of cicatricial contracture around the crater. It is seen therefore only in the chronic stage.

Appearances of the Ulcer in the Filled Stomach

The active chronic ulcer shows a crater which appears to be deep, having a narrow neck and rolled-over edges. The oedema which accompanies this stage accounts for these findings.

A fleck of barium typically remains in the crater after the stomach has largely emptied because of absence of secretory and peristaltic activity in this region.

The *Healing Chronic Ulcer* has an altered appearance resulting from (1) lessening of the oedema, making the ulcer shallower and widening its neck, and (2) the ulcer commences to fill from below.

These two changes give the healing ulcer a V shaped appearance, the angle of the V widening as healing progresses.

Indirect Signs of Gastric Ulcer.

Gastrospasm

With an ulcer on the lesser curvature, the most typical spasm is a localized notch on the greater curvature. The notch may extend around to the lesser curvature and produce a pure spastic hour glass stomach. This condition is purely a muscular spasm and may be found in cases of duodenal ulcer and is reflexly induced by biliary, appendicular and other remote abdominal lesions.

The grosser forms of hour-glass contracture are partly organic and partly spastic going on to cases of *simple cicatricial hour glass contracture* where scarring is responsible for the stenosis. The pure spastic and simple cicatricial hour glass stomach shows certain features which will help in differentiating them from carcinomatous contracture to be discussed later.

(*a*) The contracture is nearly always at the expense of the greater curvature and the isthmus is at the lesser curve. This produces a B-shape appearance.
(*b*) The lower lobe of the upper loculus is usually below the level of the isthmus.
(*c*) The point of origin of the isthmus frequently coincides with an active ulcer on the lesser curve.

To distinguish between spasm and cicatricial stenosis, medical treatment may be tried which tends to relax the spasm and reveal the nature of the narrowing. Benzedrine sulphate 10-30 mgms. may abolish spasm and should be used instead of atropine, the effects of which are less certain.

Disturbance of Pyloric Function

(1) The function of the pylorus may not be affected.
(2) It may show achalasia (Hurst) in which the pylorus relaxes infrequently, causing some delay in evacuation, but does not impede the passage of barium gently pressed into it.
(3) Pyloropasm may occur. It is a reflex phenomenon common to ulcers of the duodenum but may arise from gastric cholecystic, pancreatic, renal and appendicular lesions. The pylorus remains obstinately shut for the first fifteen minutes, simulating organic obstruction, but at some

stage of the examination normal function is demonstrated.

(4) Varying degrees of pyloric stenosis due to previous ulceration may produce (a) in the early stages, some delay in emptying, (b) later, compensatory changes, such as hyperperistalsis and gastric dilatation, (c) retention of a twenty-four hour duration which is pathognomonic of considerable narrowing of the pylorus, (d) when stenosis is so marked that almost complete obstruction is present, no twenty-four hour residue may be found because of vomiting. Dilatation, atony and almost absent peristalsis supervene.

Disturbance of Peristalsis

There may be an interruption of peristaltic waves on the lesser curve by the ulcerated area, beginning again on the distal side of the lesion. This is best seen on superimposed films taken in rapid succession. Such a "peristaltic jump" is an important sign in early carcinoma.

Then again, depending on pyloric function, peristalsis is increased at first and later diminished in pylorospasm. In gross pyloric obstruction, hyperperistalsis is the rule, but a short time after a meal, aperistalsis and atony follow.

Rate of Gastric Evacuation in Gastric Ulcer

This will depend on tone, peristalsis and pyloric function. To what has been said above may be added that a six-hour residue may be due to hypotonicity as well as to pylorospasm or to pyloric stenosis.

Localized Tenderness on Pressure

Persistent and localized tenderness on pressure over an ulcer crater is an indirect sign of ulcer, but of little importance as the crater itself is visible. Tenderness, however, does indicate an active stage of the ulcerative process. Marked tenderness over an ulcer crater is suggestive of peritoneal involvement and danger of perforation.

In absence of a crater or of deformity, the sign is uncertain and almost useless. It is significant on the lesser curvature with a corresponding incisura on the greater curvature and at points where it is difficult to demonstrate ulcer craters.

Carcinoma of the Stomach

Site. Sixty per cent arise in the pyloric region; 30 per cent in the pars media, mostly along the lesser curvature, but occasionally on the greater curve; the remainder occur in the cardiac end.

Accuracy of diagnosis by radiological methods in the hands of an expert has reached the neighbourhood of 85%.

Radiographic Classification

Scirrhous—Localized or Diffuse; Polypoid; Malignant Ulcer.

Localized Scirrhous Carcinoma

This is an infiltrative growth in the mucous and submucous layer. It affects a portion of the stomach only, in contradistinction to the diffuse type which causes leather-bottle stomach.

It produces a characteristic filling defect which may assume varied forms depending on the site involved and the extent of the growth. A common type is the napkin ring defect, as though a napkin ring were constricting the gastric lumen.

Certain radiographic features are present, common to other forms of gastric malignancy as well.

(1) Constancy in shape as observed in a series of radiograms.
(2) Aperistalsis, the peristaltic wave being traced to the upper limits of the filling defect.
(3) Lessened flexibility of the involved area.
(4) A palpable thickening.

In localized scirrhous carcinoma the outline of the filling defect is not spiky or jagged. Very early cases show no conclusive features. The peristaltic jump (described above) may be the earliest sign if the lesser or greater curvatures are involved.

In some cases the pyloric canal is affected, converting it into a rigid patent tube through which emptying is rapid.

More often the infiltration causes stenosis and mechanical obstruction.

Where the scirrhous carcinoma involves the pars media in an annular fashion, the *carcinomatous hour-glass stomach* results. As both curves are involved, the hour-glass offers a characteristic X shaped appearance. The communicating channel is usually visible in its entirety and "overhang" of the upper loculus is absent.

Where gastric capacity is curtailed, oesophageal dilatation may secondarily follow. In scirrhous carcinoma, oesophageal dilatation is not uncommon.

Diffuse Scirrhous Carcinoma (Fig. 8)

This gives rise to one form of leather bottle stomach or linitis plastica. The striking feature is diminution in size of the stomach. It presents the features described above except that the filling defect involves the entire gastric contour. Oesophageal dilatation is common and distinguishes the condition from gastrospasm.

However, simple and syphilitic forms of generalized linitis plastica present identical features and cannot be radiographically differentiated from the malignant type.

Polypoid Carcinoma (Figs. 7 and 9)

In this type of neoplasm, in addition to mucous and submucous infiltration, an irregular tumour mass projects into the gastric lumen. This mass produces a gap in the barium (Fig. 11). Smaller projections from this main mass produce rounded or oval, discrete "finger print" defects in the main gap. Ulceration taking place in these tumour masses adds further irregularity to the main filling defect in producing spiky, jagged edges. In addition the presence of a palpable epigastric mass is a common finding. Oesophageal dilatation and X hour-glass do not typically occur.

Carcinomatous Ulcers

These are usually slow growing and shallow. They are more amenable to surgical removal than the other types.

In a single examination, features suggesting malignancy may be: a size over 2.5 cms., shallowness, irregularity, variable position (i.e. those on the greater curvature are very likely malignant), absence of tenderness, and interruption of rugae without convergence.

If on medical treatment, radiological examination at the two and four week interval shows failure of the mucosal oedema to subside and the crater to shrink, the probability of malignancy arises.

Duodenal Ulcer

Site

In 95% of cases it is situated on the first inch of the duodenum. The commonest site is on the anterior wall (Clairmont), the next in order of frequency being the posterior wall.

Radiological Features may be grouped as follows:

(*a*) The visualized ulcer crater.
(*b*) Adjacent changes in the bulb.
(*c*) Secondary disturbances in the stomach.

The Visualized Ulcer Crater

The ulcer crater properly demonstrated end-on and in profile is the only pathognomonic sign of active ulceration. For the profile view, screen examination is necessary to decide the precise angle in which the radiogram must be taken. Rotation of the patient through approximately 90 degrees should then bring the ulcer face-on. Radiograms are obtained by using "aimed" exposures. To see the ulcer face-on, the bulb must be compressed to flatten it and so bring the pattern of the duodenal mucosa into relief. A central persistent fleck of barium residue marks the ulcer crater. Where oedema is marked, a translucent zone surrounds the central opacity. It is absent when the ulcer is shallow.

Adjacent Changes

When it is impossible to directly visualize the ulcer, the diagnosis is based on a persistent deformity of the shadow of the bulb (Fig. 9). The essential feature of the bulbar deformity is its constancy and

this is recognized by taking a large series of radiograms.

It must be remembered that while the deformity is most commonly due to duodenal ulceration, it may arise occasionally as a result of healed ulcers, duodenitis, cholecystitis, appendicitis, and periduodenitis.

In certain cases, pressure over the duodenal lesion produces localized tenderness. This is a variable sign. The tender spot in some cases is over the epigastrium well above the duodenal bulb. It is of no value as an independent sign.

Secondary Disturbances in the Stomach

Most frequent is hyperperistalsis. This occurring with a patent pylorus leads to rapid evacuation of the barium meal. However, pyloric function may be impaired due to pylorospasm or organic stenosis. The secondary radiological signs are of no specific diagnostic value.

The Changing Tempo of Therapeutics

"Assuming, as we must, that the only solid basis of therapeutics is clinical experience, and appreciating the many and almost insuperable difficulties in the way of educing therefrom conclusions which approximate to the 'inflexibility of arithmetic', it is an interesting inquiry, what will be the status of the therapeutical principles which have hertofore governed and of those which now govern the practice of medicine? The medical semi-centenarian has lived through radical changes in medical practice, especially as regards inflammatory affections. The so-called antiphlogistic measures—blood letting, general and local, mercurialization, active counter-irritation, emetics and cathartics, conjoined with starvation diet, which fifty years ago, were considered as indicated whenever and wherever inflammation was supposed to exist—have of late years been practically repudiated. Formerly the physicians who failed to employ them would have been considered culpable. The physician who should now resort to antiphlogistic measures, as they were formerly employed, could not expect to escape with simply the charge of fogyism; his practice would be open to censure. Now, was the practice in former days altogether wrong, and is the practice of the present time altogether right?"

Medicine of the Future
An Address by Austin Flint (Sr.), M.D.,
New York
D. Appleton and Company
1886

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THE ACUTE ABDOMEN

By JOHN A. WEDDELL, '40

The symptomatic picture, suggestive of an intraperitoneal emergency is presented to the practising physician very frequently. In order to determine whether or not the aggravating condition is intraperitoneal, intra-abdominal, or entirely outside the abdomen, a systematic investigation of the patient followed by careful consideration of the findings must be carried out in every case. To this end, a clear history, a thorough physical examination (including rectal, of course), and a few common laboratory procedures are absolutely essential to allow the investigator to answer his own mental question—"Does this patient's condition demand immediate surgical attention or does it require medical treatment?"

One must always keep in mind the numerous pathological states outside the abdomen itself, but simulating intraperitoneal or intra-abdominal emergencies. These provide too many pitfalls resulting in wrong diagnoses.

Extra-abdominal Lesions

Think of the chest first and of the troubles arising there that must be ruled out. In the **respiratory system,** early *pneumonia* may give abdominal pain with mild fever, yet the lung signs may be negligible, particularly if the pneumonic process begins centrally and there is no accompanying pleurisy. However, there is always a relatively marked increase in the respiratory rate, usually some pain or distress on bi-manual compression of the chest and sometimes slight cyanosis of the lips. True abdominal signs and rectal tenderness are absent.

Considering the **cardio-vascular system** in relation to the chest, the commonest condition suggestive of intraperitoneal pathology is *coronary thrombosis,* giving acute abdominal pain, epigastric tenderness, sometimes with slight rigidity, vomiting and collapse. There is usually a history of hypertension or previous attacks of precordial pain and one finds dyspnoea, feeble heart sounds and irregularities in rhythm, a feeling of constriction in the chest, shoulders and arms, signs of cardiac insufficiency and a falling blood pressure. An electrocardiogram provides definite evidence.

A *dissecting aneurysm of the abdominal aorta* will give a sudden, severe abdominal pain and prostration with very rapid pulse. As in coronary cases, the patient is usually over forty years of age with a hypertensive history and an enlarged heart.

Included under the heading of cardio-vascular system should be mentioned *portal thrombosis* and *mesenteric embolus,* although they are actually intra-abdominal accidents. These cases are very seldom diagnosed correctly, but give sudden excruciating abdominal pain with vomiting. The mesenteric embolus cases occur usually in patients with sub-acute bacterial endocarditis or arteriosclerosis, while the portal thrombosis cases have a history of some gastrointestinal tract infection such as appendicitis, colitis, cholecystitis, diverticulitis, or hemorrhoids.

A few rare cases of *acute pericarditis* have been confused with an acute abdominal condition but the auscultatory finding of a pericardial friction rub is diagnostic.

In consideration of the **nervous system** next, the commonest mistakes are made with a patient suffering from *lead colic,* who is having crampy abdominal pain. The occupational history, the blue line around the gum margins, the stippling of the red blood cells, the loss of weight and thumb movements are all indicative but the immediate relief after 5 cc. of 10% calcium chloride solution intravenously, clinches the diagnosis.

Acute meningitis at its onset will give abdominal pain, vomiting and fever but there is also pain in the extremities as

well as signs of meningeal irritation—positive Kernig and Brudjinski tests.

Gastric crisis of tabes, with acute abdominal pain, vomiting and rigidity is now quite rare. The Argylle-Robertson pupil and the absent ankle reflexes indicate the nature of the condition. Pain and rigidity from involvement of spinal nerve roots by injury, chronic infection or new growth should not be mistaken as there is absence of other abdominal signs and the pain is aggravated by movements of the spine.

Certain **metabolic conditions** may give rise to abdominal symptoms. These are *exophthalmic goitre*, *acute acidosis of diabetes*, *hypoglycemia*, and *acute uremia* and in each the diagnosis should be evident from the characteristic findings.

Intra-abdominal Lesions

The **urinary tract** is intra-abdominal but not intraperitoneal. *Renal colic* causes spasms of terrific pain, felt down the same side of the abdomen to the groin and genital region and is sufficient to cause the patient to writhe in pain. The temperature is normal while the pulse is elevated during the spasms, returning to normal in the interval.

Sudden distension of the pelvis of the kidney will result in severe pain down the same side of the abdomen, followed by hematuria and albuminuria.

Acute pyelonephritis begins usually with fever and chills, pain in the upper quadrant of that side and around to the back. There is costovertebral tenderness and the urine shows a large number of pus cells and usually some blood and albumin.

Acute prostatitis and *acute urinary retention* will both give lower abdominal pain, but there is usually perineal distress, a history of frequency, dysuria and retention, and in the case of the latter, the bladder is palpable suprapubically.

Lesions in the **genital tract** produce symptomatology very similar to that with intraperitoneal involvement, but the problem of differentiation arises almost entirely in the female sex. With lower abdominal pain in a woman, one should always keep in mind the possibilities in relationship to pregnancy. *Early pregnancy* gives nausea, mild pain and vomiting, while on towards term *accidental hemorrhage* may be confused with *appendicitis*. The commonest condition mistaken for appendicitis is *acute salpingitis*. These women, however, usually have some recent history of exposure to gonococcal infection, some form of instrumentation or a recent labour or abortion, as well as painful prolonged menstruation, indicative of pelvic inflammation. The onset is usually in the lower abdomen—an intermittent, mild pain increasing gradually in severity and becoming steady in character. Tenderness is felt, sometimes on one side but often on both sides or else towards the midline. Vaginal examination reveals a white discharge and definite tenderness mostly in the posterior and lateral fornices. It is well to remember that an acute appendix in a pelvic location can cause a salpingo-oophoritis.

Tubal pregnancy must also be considered as a possible cause of abdominal symptoms in a woman. It in itself can give vague lower abdominal pain, often crampy in character, but more severe results occur with a tubal abortion, or with a ruptured tubal pregnancy. There are many of the characteristic signs of pregnancy such as softening of the cervix and enlargement of the breasts and there may or may not be disturbances in menstruation. The pain, tenderness and palpable mass are on the same side, and vaginal examination usually shows some bleeding, the presence of the mass and tenderness in all fornices if rupture or abortion have taken place.

A *ruptured Graafian follicle* is often very confusing particularly if from the right ovary. There is sudden pain, low in the abdomen on one side, occurring at the mid-menstrual point, the pulse and temperature are normal and there is no palpable mass or discharge. Sometimes this condition cannot be distinguished from acute appendicitis.

Cysts of the ovary will give mild lower abdominal pain and tenderness to palpation. On examination of the abdomen, a cystic mass can usually be felt and there

is often evidence of free fluid. Torsion of the pedicle of an ovarian cyst will cause a sudden severe lower abdominal pain with rigidity of the muscles, moderate pyrexia and leucocytosis, and the accident results from severe exercise, in most cases. There is evidence of a tense, tender mass in the lower abdomen. Occasionally in the male, torsion of the testis occurs with severe lower abdominal and perineal pain but the localization of the cause is not difficult.

Intraperitoneal Lesions

In the consideration of the actual intraperitoneal conditions that are suspected in a patient with symptoms referable to the abdomen, it is well to recall that all true emergencies here result from one of three basic causes namely—acute inflammation, haemorrhage, and obstruction. Dr. R. R. Graham has stressed three cardinal findings indicative of intraperitoneal involvement—associated pain and tenderness, one point of maximum tenderness, and thirdly, both pain and tenderness aggravated by rolling over in bed.

The commonest condition met in practise is *acute appendicitis*, which, if unrecognized takes one of four courses, namely resolution, formation of an appendiceal mass, formation of an appendiceal abscess, and lastly general peritonitis. A typical case begins with mild crampy distress, usually peri-umbilical in location, but frequently across the lower abdomen and occasionally in the epigastrium. After six to twelve hours the pain becomes localized as a steady distress in the right lower quadrant, and physical examination reveals localized tenderness, hyperaesthesia, and often rigidity. There is definite rebound tenderness, the temperature is 100 to 101, with slightly elevated white blood count in most cases. However, the picture is quite variable and may actually be quite atypical. If the appendix is low down in the pelvis it will not usually show abdominal findings but it is in this type of case that the finding of tenderness on rectal investigation is most useful. In the obstructive case, where a fecolith has lodged in the lumen cutting off the blood supply, the appendix can become gangrenous within a few hours, and without showing the characteristic clinical stages. Treatment consists in removal of the appendix at once unless there is evidence of abscess formation or development of peritonitis, in which case conservative treatment is followed and the appendix removed at a later date.

Acute enterocolitis is a very common complaint and is of no serious import other than to recognize it as a case for medical, not surgical care. This acute catarrhal inflammation of the mucous membrane of the small or large intestine may be due to bacteria, decomposed food products irritative in nature or an allergic response to certain foods. The onset is slow, with lassitude, nausea, colicky pain, diarrhoea and sometimes tenesmus if the rectal mucosa is involved. There is a mild fever of 100-101, with considerable thirst. The abdomen shows no localized pain or tenderness but is often diffusely tender to palpation. The symptoms subside in a short time varying from a few hours to a few days.

Acute cholecystitis is the next most common condition in this group. There is sudden onset of pain in the right upper quadrant of the abdomen, often referred to the shoulder of the same side, the epigastrium or the back. Nausea and vomiting may occur and if so, always follow the pain and do not precede the onset. There is moderate pyrexia of 101-102, moderate leucocytosis and some embarrassment to respiration. The gall-bladder may be enlarged and tender and there may be slight rigidity in the right upper quadrant, and sometimes an area of epicritic hyperaesthesia can be demonstrated one inch or so from the vertebral column in the costo-vertebral angle. If gall-stone colic is part of the picture, the pain may be very severe, coming on in spasms in the epigastrium and felt in the back as well, but passing off quickly. Although there are two opposing schools of thought in regard to the procedure indicated in the treatment of acute cholecystitis, the conservative method is the accepted one in this city at the present time.

Intestinal obstruction is probably the

next most commonly found intraperitoneal condition. The onset is marked by rhythmical colicky pains in the abdomen with accompanying nausea, the pulse and temperature being normal. Later, vomiting occurs, and as the condition progresses there will be some distension after a sufficient period of time has elapsed providing the point of obstruction is not too high in the small bowel. Fluid levels will then be visible due to separation of the gas and fluids in the bowel. In small bowel obstruction, vomiting is more marked but distension is less than in the large bowel where most obstructions occur at a later age period, usually beyond forty. The procedure indicated is to relieve the obstruction, reduce the distension and restore the biochemical balance of the patient. In small bowel obstruction, the biochemical upset is more marked, as the vomiting promotes extreme dehydration and the temperature and white blood count are both elevated. In this group one should mention in particular, *strangulated femoral and inguinal herniae.*

Acute perforated ulcer of stomach or duodenum occurs with sudden agonizing epigastric, pain, often in a patient with no previous history of ulcer. The patient is pale, anxious and sweating but his temperature remains normal or even subnormal. There is marked rigidity of the upper abdominal wall and usually absence of liver dullness. The temperature and pulse are normal and there is no vomiting. There may be rectal tenderness due to fluid from the perforation having run down into the right iliac fossa. A latent period with absence of signs and symptoms may follow the onset in some cases and be very misleading. Laparotomy and repair of the perforation are always indicated.

Internal hemorrhage is a condition frequently met and occurs from the ovary or Fallopian tube in the female giving signs and symptoms of acute lower abdominal involvement with internal bleeding. The patient is restless, becomes pale, and the pulse and respiratory rates are elevated, along with the white blood count. There is absence of any vomiting or rigidity but usually some cutaneous hyperaesthesia of the lower abdominal wall is present. Vaginal examination reveals some vaginal hyperaesthesia and a boggy mass in the pouch of Douglas.

Acute Pancreatitis is one of the less common conditions, and is considered here although it is not a structure which is actually intraperitoneal. It occurs in an older person who has had, in most cases, some previous pathology of his biliary system. There is severe epigastric pain felt through to the back, but absence of rigidity. Deep epigastric tenderness can sometimes be elicited and also tenderness in the left costo-vertebral angle. Some cases show localized patches of discoloration of the skin of the loins. Usually a laparotomy must be done to verify the diagnosis but if one is sure of the diagnosis, conservative treatment is indicated.

Acute colonic diverticulitis is another less common condition and in its symptomatology, suggests a left sided appendicitis, with localized associated pain and tenderness in the left lower quadrant. A mass is palpable in about 30% of the cases, and there is usually rebound tenderness. As in appendicitis, the danger here is the development of peritonitis from rupture of the inflammed diverticuli. If conservative treatment is of no avail, a colostomy can be done with intention of repair in eighteen months time if the condition is sufficiently improved.

Regional Ileitis is a low grade inflammation of the lymphoid tissue of the ileum, It becomes granulomatous in nature and causes lymph blockage. Clinically there are several stages, first merely some abdominal discomfort and slight tenderness, then more marked pain and diarrhoea after a few month's time. Subacute obstruction occurs with colicky pain and nausea, and finally if the condition is not treated, adhesions and fistulae may develop. The usual procedure for relief is a resection of the involved portion of the gut although recently some men claim results from temporary colostomy.

Disturbances in nervous control of portions of the bowel are rare conditions. *Paralytic ileus* with signs of obstruction

is one form of disturbed mechanism, due either to infection, as in peritonitis, or to trauma as in a bullet wound involving the abdominal wall. There are occasional rare cases of *acute dilation of the stomach,* usually occurring postoperatively with regurgitation, epigastric pain and dehydration.

Rare cases of *torsion* of such structures as the *gall-bladder* or part of the *omentum* have occurred with sudden severe abdominal pain and absence of signs of infection, or hemorrhage where laparotomy is necessary for relief.

Psychological Aspects of Cod Liver Oil Administration

Some authorities recommend that cod liver oil be given in the morning and at bedtime when the stomach is empty, while others prefer to give it after meals in order not to retard gastric secretion. If the mother will place the very young baby on her lap and hold the child's mouth open by gently pressing the cheeks together between her thumb and fingers while she administers the oil, all of it will be taken. The infant soon becomes accustomed to taking the oil without having its mouth held open. It is most important that the mother administer the oil in a matter-of-fact manner, without apology or expression of sympathy.

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FOOD - TONIC - BEVERAGE

CARCINOMA OF THE STOMACH

By R. M. HALL, '41

The problem of cancer is an ever increasing one. This disease was the third commonest cause of death in Canada in 1938. The stomach is probably the most frequent site of cancer in the male, as is the uterus in the female, and furthermore, the United States Census of 1912 showed a more rapid increase in cancer of the stomach than of either uterus or breast.

The geographical pathology is also interesting. It has been shown that there are marked variations in different countries and cities; negroes and those living in tropical countries showing a slight relative immunity.

Males are more often affected than females in the approximate ratio of 10:7, the total number of deaths being approximately 2% of the deaths from all causes.

There are several important factors recognized in the aetiology of this disease. The average age incidence is 61 years, the greatest number of deaths occurring between the ages of 40 and 70. However, there is noted a rather frequent occurrence between 30 and 40, and even between 20 and 30, while Cullingworth has reported one case of congenital adenoma of the pylorus in a 5 weeks old infant. Those occurring in the third decade tend usually to run a rapid course.

Heredity is another important aetiological factor. Welch found in 14% of 1,744 cases of gastric cancer, that other members of the family suffered from cancer. Manishon reported 23 families with 69 cancerous members of whom 57 had stomach cancer. From such collections of data it is possible to assume a tissue predisposition, but the rarity of these observations emphasize the usual absence of any element of heredity in gastric cancer.

Trauma is not considered to be an important aetiological factor by most observers.

Idiosyncrasy as a factor is mentioned by R. Schmidt, who describes two types of subjects in whom gastric carcinoma is prone to develop. His "dynamic type" is a well-nourished individual, whose parents were long-lived and whose digestion had always been so good that they may be called "gastric athletes". These people develop the disease in later life. His "adynamic type" is one who is poorly nourished and phthisic, who suffers from gastric ulcer or achylia. These develop the disease early. Alcoholism was noted in 15% of cases by Friedenwald.

Carcinoma of the stomach occurs in the pyloric region in 63% of cases, in the lesser curvature and cardia in 20%, the remainder being in the walls of the greater curvature. It is interesting to compare the distribution of carcinoma with that of peptic ulcer. Approximately 60% of gastric cancers are located at the pylorus, while 12% of ulcers occur in this region. Ulcer occurs on the lesser curvature in 36.3% and cancer in 12.2%. Boyd states that about 5% of chronic gastric ulcers may develop into carcinoma. It is probable that not more than 2.5 or 3% become malignant, and that higher estimates by various observers are due to difficulties in interpreting the presence of irregular epithelial structures in the edges and submucosa of an ulcer. There is considerable disagreement as to whether or not epithelial structures in the submucosa mean carcinoma.

Pathology

Carcinoma of the stomach appears in several characteristic anatomical forms, the microscopic appearance being somewhat different in the various forms.

Papillary Type. In this form there is a circumscribed polypoid or fungating growth, which eventually ulcerates causing infection and haemorrhage. This tumour may reach very large dimensions before invading the regional lymph nodes, but is more malignant in the stomach than

in the uterus or colon, where similar tumours occur.

Excavating Type. This is a more common lesion. It is only slightly elevated and ulcerates early. These are found most commonly in the region of the pylorus, and probably develop in some cases from a chronic gastric ulcer. The diameter is usually greater than 2.5 cm., the cut surface being considerably thickened and showing yellow flecks of necrotic tissue. The surface is nodular.

The microscopic appearance of these growths is that of an adenocarcinoma, presenting various imitations of a gastric tubule, which are usually of an orderly nature. The original tubules become much elongated, bifurcated, and sacculated. The tumour consists of large numbers of such alveoli separated by stroma which is usually scanty. The individual cells are cylindrical or cuboidal with clear cytoplasm and hyperchromatic nuclei. When these tumours infiltrate or metastasize, the alveoli are usually lined by single rows of cells, while similar tumours of the colon more often present multiple layers of large cells. The tumour cells penetrate the muscularis mucosa and spread widely through the submucous coat, finally appearing on the serous surface.

Mucous changes are frequently seen in gastric cancers, especially in the adenocarcinomatous types. When this change becomes pronounced, it alters considerably the gross appearance of the growth. Characteristically this change begins in the pyloric region and infiltrates all the coats of the stomach wall, which may now measure 2-3 cm. in thickness. The stomach as a whole may be reduced in size, but not as much as in the true scirrhous type of carcinoma. The mucosa, submucosa, and the muscularis mucosa become largely replaced by a gelatinous material which also appears in the form of translucent nodules on the serous surface. Adhesions to neighbouring organs are frequent in this type of growth, and invasion to neighbouring lymph nodes is common, but late. Colloid visceral metastases are rare.

Diffuse Medullary Type. These tumours produce two well-defined anatomical forms. The gross appearance may be that of a soft, rounded, more or less circumscribed mass of considerable extent, which often ulcerates, and which appears most often in the region of the cardia or the fundus. These growths are frequently of a telangiectic type, thus being subject to severe haemorrhage. Fatty degeneration and necrosis lead to widespread ulceration and perforation, or erosion of arteries. There is frequent involvement of neighbouring organs, and early metastases to distant ones, indicating a high degree of malignancy.

A more diffuse type may arise from any part of the gastric mucosa, infiltrating all the layers of the stomach wall to such an extent that the original tumour is hard to locate. These tumours commonly arise in the pyloric region, and when ulceration occurs, pyloric stenosis and marked dilatation of the stomach results. Perforation is also common in this type. It is also of a high degree of malignancy, showing early involvement of lymph nodes and distant metastases.

Microscopically these tumours show an anaplastic type of cell, usually with some attempt at formation of alveoli, which may be large or small. The cell type is atypical, the cells are usually small polyhedral or round, with relatively large, hyperchromatic nuclei. Different formations may be seen in different parts of the tumour. In some places, attempts at formation of glandular acini are seen, in other places, dense masses or columns of cancer cells are found, separated by a dense stroma of the scirrhous type. These tumours are sometimes mistaken for lymphosarcomas, due to the complete lack of desmoplastic quality.

Linitis Plastica—(Diffuse Infiltrating Carcinoma, Cirrhosis of the Stomach or Leather Bottle Stomach). Here there is usually involvement of the whole stomach, which is very small and thick-walled. The normal stomach is about 12 inches long and has a capacity of about 40 ounces, but the stomach which is the seat of this type of cancer is perhaps only 4 inches long and holds only 4 ounces. The whole wall of the stomach, starting in the

pyloric region is thickened in all its coats, especially in the submucosa and muscularis mucosa. It is peculiarly thick and rigid. There is very seldom ulceration of the surface, but the mucosa is firmly glued to the underlying muscular coat. The serosa is opaque, fibrous, and covered with small nodules from invaded lymphatics. There may be adhesions to neighbouring organs. There is regular involvement of the lymph nodes, and large metastases may be found in the liver and other organs.

Microscopically the majority of scirrhous carcinomas present small or large groups of polyhedral cells. However, as the fibrosis increases, the tumour cells diminish, and large areas of fibrous tissue may be seen containing only small groups of degenerating cells, or no cells at all. The neighbouring nerve trunks, lymphatics and capillaries may show much larger and more typical cells. The secondaries in the lymph nodes and peritoneal nodules may show typical tubular growth.

There is, in cancer of the stomach, a change in the gastric mucous membrane, not only in the region of the tumour, but also at a distance. There are inflammatory or atrophic changes, an interstitial gastritis with atrophy of the peptic cells and tubules, and a metaplastic transformation of gastric into intestinal type of cell lining, the goblet cells being increased in number. There is an increase in the secretion of alkaline mucus which tends to neutralize the gastric acid, thus causing an early disappearance of free gastric acid.

After the carcinomatous focus has been established, the mode and rate of extension depends chiefly upon the growth energy of the cancer cells, so that there are wide variations in the behaviour of the different tumour types.

Local spread. This occurs in the stomach wall and to neighbouring organs, causing adhesions. The adenocarcinomas begin in the mucosa but perforate early into the submucosa, where they spread readily through the loose tissue and along the lymphatics to the lymph nodes. The cancer cells may spread to the serous surface, drop off, spreading throughout the whole peritoneal cavity by implantation, causing a generalized carcinomatosis. The duodenum is seldom invaded.

Lymph spread. This is extremely common, especially in the case of the diffuse types, where early spread occurs by means of the lymphatics in the submucosa. The first lymph nodes to be involved are the regional ones of the stomach, whence the cells spread along the thoracic duct and other lymphatics to the supraclavicular, perigastric, diaphragmatic, and thoracic nodes. The importance of an enlarged supraclavicular gland in the diagnosis of abdominal cancer, and especially gastric cancer is well known.

Blood spread. This is the method by which distant organs become involved. The liver is usually the first organ invaded by way of the portal vein. The lungs, central nervous system, kidneys and bones are involved in the order given. In the ovary, the characteristic Krukenberg tumour may be secondary to gastric carcinoma. Two interesting cases of spread to bone may be cited from the *New England Medical Journal.* In one case from a primary in the stomach following peptic ulcer, every inch of vertebral and sternal marrow was taken up with metastases. In a second case of undifferentiated gastric cancer following an ulcer, there was widespread invasion of bone marrow, the secondaries showing definite gland formation.

Symptomatology

A knowledge of the signs and symptoms of gastric cancer is very important, as early diagnosis and treatment is the only method of cure. The initial symptoms may be vague and variable. The previous digestive or gastric history is almost unknown except in those cases in which carcinoma follows a chronic peptic ulcer, of several years duration. Two groups of incipient cases present themselves for diagnosis. In one there is a definite history of gastric disturbance. In the other the disease appears in a perfectly healthy subject. Many patients date their illness back to a specific dietetic

error, trauma, or an acute disease. Long-continued abuse of the stomach is commonly overlooked until the disease reaches a stage when it produces symptoms, which once established, are usually persistent.

The persistence of a feeling of oppression which has an acute onset after meals distinguishes cancer from a simple functional disturbance. Pain is a very important early symptom, being often of a type which is influenced by diet. It appears one or two hours after eating or when the stomach is empty, is often worse at night and may be aggravated by exertion. Loss of weight is sometimes an early symptom, but may be delayed. Regurgitation is an early symptom, which may soon be succeeded by vomiting.

Diagnosis

If a patient over forty years of age, complains of vague digestive symptoms such as discomfort after meals, diminution in appetite and slight loss of weight, it is essential that this person be considered to have cancer of the stomach until proven otherwise by means of a thorough physical, radiological and laboratory examination. In many cases the symptoms and the history will be more marked, and the diagnosis easier, but it is the healthy, "dynamic" individual who develops the disease insidiously, and requires thorough examination for further signs.

The main signs of carcinoma of the stomach which should be investigated are:

(1) Careful palpation of the epigastrium may reveal a lump, the position and nature of which must be carefully studied.

(2) The presence of occult blood in the stool, determined by means of the benzidine test. If gross blood is found in the stool, the bleeding is probably from the colon or rectum.

(3) Repeated radiological examination. After suitable preparation, the patient is given a barium meal, and radiographs are made. These must be examined with special attention to any filling defects, diminution in the function of the pylorus, or changes in the motility of the stomach.

(4) The presence of lactic acid and the decrease or absence of free hydrochloric acid, determined by chemical analysis of the gastric contents, may be early findings in carcinoma of the stomach, but are not constant, and may be present in other diseases. Also, when cancer is superimposed on a gastric ulcer, the hydrochloric acid may be increased, not diminished.

(5) Presence of tumour fragments in the vomitus discovered by microscopic examination, is a sure sign.

(6) Gastroscopy may locate early lesions of the cardia and of the greater curvature. A biopsy may be done on a suspicious ulcer.

(7) A blood count may reveal a well-marked secondary anaemia, which is a very constant occurrence in carcinoma. The anaemia may be occasionally indistinguishable from Pernicious Anaemia, except that the icteric index is not above normal. A leucocytosis may also be present.

There may be a reduction in the motor power of the stomach, so that the passage of its contents into the duodenum is delayed. This is due to a chronic interstitial gastritis. In pyloric carcinoma, a tumour which is palpable above and to the right of the umbilicus, and is at first movable, later becomes fixed. It may receive pulsations from the aorta. The pylorus becomes contracted, so that the stomach dilates and the greater curvature is displaced downward, perhaps almost into the pelvis.

In the later stages, pressure symptoms manifest themselves in all types of cancer of the stomach. Ascites from compression of the portal vein, jaundice from implication of the common bile duct, oedema of the legs and varices of the superficial abdominal veins from pressure on the inferior vena cava, are common. The peritoneal cavity may be distended with fluid. A palpable left supraclavicular lymph gland is also common.

A Clinical Example

A male of 43 years of age gave a history of duodenal ulcer which changed two years after his first complaint, to one of pyloric obstruction with vomiting. He had lost 45 pounds in the year preceding his death. Physical examination

revealed a large mass in the epigastrium. A laparotomy was carried out two months before death and a diagnosis of inoperable carcinoma of the stomach was made. He gradually went downhill, and died two years after the onset of his symptoms of pyloric obstruction.

Autopsy revealed a large colloid carcinoma of the stomach which had caused pyloric obstruction. A few secondaries in the immediate neighbourhood of the stomach were found. No duodenal ulcer was demonstrated at autopsy. The immediate cause of death was bronchopneumonia of the lower lobe of the left lung.

Treatment

At present, surgery offers the only hope of cure for carcinoma of the stomach. It is estimated that of selected operable cases, about 20% may be cured surgically. For cancer of the pylorus, if the mass is comparatively movable, removal of the diseased portion, together with the lymph glands along the greater and lesser curvatures, may be undertaken. When the growth is firmly adherent, gastroenterostomy may be required for food passage. If the cancer is in the body of the stomach, a partial or total gastrectomy may be feasible in the absence of massive adhesions. If this is impossible, and there is evidence of obstruction to food passage, a gastroenterostomy is undertaken. Usually the anterior operation is performed, due to cancer of the posterior wall.

Radium and deep X-ray therapy have proved to be extremely disappointing, although in rare colloid carcinomas such treatment may arrest the disease temporarily. Treatment by drugs is purely symptomatic, but is none-the-less very important as a palliative measure.

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PERITONITIS

By T. H. BLACK, '41

Peritonitis is a subject which a paper of this scope cannot hope to cover in any detail. It is usually the complicating factor or the end result of an underlying pathological condition, the diagnosis of which, granted that the case is seen in time, should obviate the necessity for the diagnosis of peritonitis.

In this paper we shall attempt to describe acute peritonitis as it arises secondarily and discuss somewhat more briefly the primary and rarer condition.

Etiology

Acute peritonitis is the result of the presence of bacteria or some non-septic irritating substance in the peritoneal cavity. The bacteria most commonly found are B. coli and Streptococci, followed by Staphylococci, Gonococci, Pneumococci, B. pyocyaneus, B. typhosus, the Tubercle Bacillus and certain anaerobes. The infection from the Pneumococcus and Tubercle Bacillus will be treated separately later.

Non-septic peritonitis arises from haemorrhage into the peritoneal cavity, the flooding of the membrane by the contents of a ruptured cyst, the presence of a foreign body such as a sponge left in post-operatively, or the presence of some irritating chemical such as that used in the non-operative treatment of hernia.

Bacteria reach the peritoneal cavity in three ways:

(1) *From an abdominal organ.* Usually the perforation of a hollow viscus is the cause; appendicitis is the commonest. Others are gastric, typhoid or dysentery ulcers or perforation in a carcinoma. Obstruction untreated is another cause, the bacteria in intussusception, volvulus, or complete stenosis, finding their way through the wall of the gut which is either gangrenous or inflamed and oedematous.

The female pelvic organs, particularly in puerperal sepsis, are another source. The infection spreads by direct extension through the uterine wall or by instrumental perforation in attempted abortion. Gonococcal Salpingitis frequently gives rise to a local or diffuse pelvic peritonitis depending on whether the purulent material escapes via the ostium or not.

(2) *From the exterior.* This may result from an accident as in wounds from shrapnel or bullets, the piercing of the abdominal wall in accidents of civil life or from faulty technique in surgery.

(3) *Hematogenous.* These are rare but are seen in some pneumococcic, tuberculous and streptococcic septicaemias. Primary streptococcic peritonitis developed in four out of 3,670 cases of scarlet fever in one series published from the Riverdale Isolation Hospital.

One form of non-septic peritonitis which causes difficulty in differential diagnosis from acute appendicitis is that benign form which occasionally results from the rupture of an ovarian follicle.

Pathology

Peritonitis may be local or diffuse, the former being the commoner. Boyd gives a very precise description:

"The membrane appears first pink and injected. Then the sheen is lost and is replaced by a frosted appearance due to the formation of a layer of fibrin on the surface. Finally, cords of bowel are glued together by a sticky exudate. Meanwhile a fluid exudate forms and collects especially between the adherent coils of bowel. At first, it is serous but soon becomes purulent. . . . The microscopic appearance is that of an inflamed serous membrane. The chief element of the exudate in the early stages is fibrin, but if the condition becomes purulent, there may be a thick layer of polymorphonuclear leucocytes together with a variable number of red cells. . . ."

Spread is by lymphatics or the surface. In surface spread the greater omentum plays an important part, travelling to the local site and walling it off by adhesions. Lymphatic spread usually occurs in streptococcic infections.

In most cases the peritonitis is localized by adhesions between bowel or omentum and the parietal layer. Abscess formation may take place at the site of infection or may trek via the gutters of the posterior abdominal wall to form a subdiaphragmatic abscess.

Diagnosis

This may be difficult in the early stages since the signs are usually masked by those of the underlying lesion. It is often difficult to tell at what stage of the process one set of symptoms start and the other leaves off. There are certain classical signs and symptoms. The recognized symptoms are:

(1) Suddenness of onset
(2) Pain
(3) Vomiting
(4) Bowel dysfunction
(5) Anxiety
(6) Prostration.

The common signs are:

(1) Posture and expression
(2) Dry tongue
(3) Rise of temperature and pulse rate
(4) Leucocytosis
(5) Splinting of the abdominal muscles
(6) Rebound tenderness
(7) Hyperaesthesia
(8) Peters' sign

Acute peritonitis begins with the sudden onset of acute abdominal pain usually near the site of the original lesion. The pain is of a boring, burning type; continuous, with excruciating exacerbations. With septicaemia, the pain subsides.

Vomiting occurs shortly after the onset, emptying the contents of the stomach, and is repeated subsequent to attempts to feed by mouth. Later, if ileus occurs, the vomitus takes on the character of that found in intestinal obstruction.

Diarrhoea may occur shortly after onset followed by complete constipation.

The patient feels acutely ill and anxious, as if sensing disaster. Although alert, he is extremely weak and prostrated.

He lies on his back with thighs flexed on the abdomen, breathes through his thorax and avoids adventitious movement as much as possible. His eyes are bright and enquiring; his face is pale and drawn. Pulse and temperature are up. A temperature of 102 degrees Fahrenheit is not uncommon.

Leucocytosis from 15,000 to 20,000 is usually present with a great increase in polymorphs.

On palpation the abdomen is rigid, the recti splinted. This may be localized or general.

By far the most important sign is that of rebound tenderness where the tenderness is felt, as pressure on the abdominal wall is released.

Locally there may be an area of hyperaesthesia and the localized mass of an abscess may be felt.

George Armstrong Peters, Professor of Surgery in this University at the beginning of the century, described a sign wherein the heart sounds are plainly heard over the abdominal wall in acute appendicitis.

By and large, the onset of acute abdominal pain with vomiting and rebound tenderness spells a diagnosis of acute peritonitis.

Treatment

Surgery is usually indicated. In fifty consecutive cases of acute appendicitis complicated by localized peritonitis, treated at the Toronto General Hospital from 1927 on, twenty-six were treated by drainage alone, thirteen by appendicectomy with drainage and the rest by conservative treatment. Conservative treatment was by local heat and fluids.

Death occurred in six cases. Five of the deaths were treated by drainage, three complicated by other extensive surgery, and one, a male age 64, with a three weeks' history of pain. One death was treated by appendicectomy and drainage

but a duodenal ulcer resection was done at the same time. In all the deaths the time from the beginning of symptoms to diagnosis and treatment varied from ten to twenty-one days. If, from such a short series, any conclusions can be drawn, it would appear that the best risks are those of people in the second and third decade diagnosed within two or three days of the onset of symptoms and treatment instituted to relieve the emergency alone.

In cases of primary streptococci infection it would seem that sulphanilamide were the best treatment. In cases reported, the best results were obtained where the drug was injected into the peritoneal cavity following paracentesis. In this series, the only deaths occurred where further surgery had been done. Sulphanilamide has changed the prognosis also in cases of secondary streptococcic peritonitis.

Prognosis

Prognosis depends on varying factors. The age of the patient, duration of symptoms before treatment, the presence or absence of paralytic ileus and the judgment of the surgeon. Toxic absorption may play a part in the morbidity of the patient but he dies from paralytic ileus.

Pneumococcal Peritonitis

This may be primary or secondary. The primary form is usually found in young female children from three to seven years of age and enters the peritoneal cavity by way of the genital tract. It begins as a pelvic peritonitis and spreads rapidly. The secondary form occurs as a complication in pneumococcic pneumonia or otitis media and develops either by direct extension through the diaphragm or by the hematogenous route.

The exudate is purulent and there are no preceding abdominal signs.

Treatment in the past has been by drainage administration of serum but sulphanilamide is reported to be very successful in these cases.

Tuberculous Peritonitis

This type may be acute or chronic, profuse or local, and plastic or with effusion.

The sources are from tuberculosis of the bowel, mesenteric glands, Fallopian tubes, or as is generally the case, from lungs or pleura. There may be an hematogenous spread from bones and joints.

In the acute form, there is difficulty in differentiation from the ordinary acute peritonitis. Usually, tuberculosis in some other form is present, and the moderate leucocytosis is characterized by a relative increase in monocytes.

The chronic form may result from an acute attack or may be insidious as when spread from the ileo-caecal region occurs. In the wet form the abdomen is distended by fluid—an exudate of a specific gravity greater than 1.018 and albumen greater than 3. It contains lymphocytes and may be blood stained. The peritoneum is studded by tubercles. In the dry form a plastic fibrinous exudate forms which glues the bowel together. It may be recognized by the signs of obstruction developing in a pulmonary case or the formation of fistulae.

In children, the first sign is often a transverse tumor about the umbilicus formed by the tubercle-studded rolled up omentum.

Treatment:

Surgical intervention is of aid in removing the focus of infection. In cases of peritonitis complicating pulmonary tuberculosis there is often an unexplained result from laparotomy. Though nothing is done beyond the incision, the condition clears up.

When a great number of adhesions are present, little can be done for the relief of symptoms.

Left to itself, the disease may become chronic or clear up. Aspiration of the ascitic fluid in the wet form gives relief. General measures in the treatment of tuberculosis are indicated.

Though the prognosis looks bad, one should not despair in one's outlook, for untreated cases are often surprising in the extent of their recovery.

TRAUMATIC NERVE LESIONS OF THE UPPER LIMB
WITH THEIR ASSOCIATED CLINICAL SIGNS

By C. J. ROBSON, '42

The nerve supply to the upper limb is derived from the anterior primary rami of the lower four cervical nerves and the greater part of the anterior primary ramus of the first thoracic nerve. The sensory supply to the medial side of the arm is via the intercosto-brachial nerve (Th. 2), and a varying degree of sensation over the shoulder by the supraclavicular nerves. (C-3, 4.)

The five nerve roots unite in the posterior triangle of the neck to form the brachial plexus which passes behind the clavicle to enter the axilla where the cords of the plexus surround the second part of the axillary artery. In the lower part of the axilla the plexus splits into the nerves for the upper limb.

Injury to the nerve supply of the limb may occur therefore as a result of:—

(A) Injury to the roots of the brachial plexus and to the plexus proper.

(B) Injury to the terminal nerve divisions of the plexus.

Injury to the Brachial Plexus

The brachial plexus may be injured as the result of accidents in which there is a sudden wrenching of the limb as when a person attempts to grasp something when falling. The nerve roots are wrenched from their attachments or the nerve trunks may be compressed against the first rib by the clavicle. The plexus is also found to be injured as a result of fractures of the clavicle and dislocations of the head of the humerus, the latter resulting particularly in injury to the medial cord. In infants, a birth palsy is occasionally seen in which the roots of the plexus are stretched or torn as a result of too much traction on the head during delivery.

Clinical Signs:—These may conveniently be divided into those resulting from

(1) Paralysis of all the nerve roots.

(2) Paralysis of only some of the roots.

(1) If *all the nerve roots* are involved the whole limb is paralyzed and lies flaccid and anaesthetic by the side. Sensation is present down the medial side of the arm as far as the elbow since this is the area supplied by the intercosto-brachial nerve. Paralysis often involves the pectoralis and scapular muscles but the rhomboids and serratus magnus retain their nerve supply. Since there is a contribution from these nerve roots to the sympathetic system, oculo-pupillary changes are seen if these fibres are involved in the lesion, viz.:—the pupil is contracted and mydriasis is defective on the exclusion of light.

(2) *Incomplete paralysis* may involve cervical roots 5 and 6 producing an upper arm or Erb-Duchenne paralysis in which loss of power is found in the deltoid, spinati, brachialis, biceps and brachio-radialis muscles as well as the clavicular head of pectoralis major, latissimus dorsi, teres major, rhomboidei and subscapularis. The arm lies close to the side and cannot be abducted or adducted. A certain degree of medial rotation usually appears after a lapse of some time, especially in children. The forearm is extended and in a position of pronation, supinating power is lost and the elbow cannot be flexed. Movements of the hand and wrist are usually unaffected, but occasionally the wrist flexors are paretic.

If the lesion is confined to the roots of the last cervical and first thoracic nerves, the resulting paralysis is of the lower arm or Klumpke's type. The flexors of the wrist and fingers as well as the intrinsic muscles of the hand are paralyzed, with the result that the hand assumes the claw shape which is similar to that found in paralysis of the ulnar nerve and will be

described under that nerve. The area of cutaneous sensation which is affected is also similar.

Injury to Nerves

Circumflex Nerve. This nerve, due to its intimate relationship with the surgical neck of the humerus, is vulnerable to such injuries as result from a blow on the shoulder, dislocation or fracture of the upper end of the humerus, pressure due to poorly constructed crutches, etc.

Clinical Signs:—The deltoid and teres minor muscles are paralyzed. The paralysis of the teres minor is not easily demonstrated, however. Due to the deltoid paralysis, the arm cannot be raised or abducted from the side of the thorax and an attempt to raise the arm leaves the fibres of the deltoid loose, a point which immediately rules out ankylosed shoulder joint. The deltoid region atrophies and the acromion becomes prominent.

Musculocutaneous Nerve. A paralysis limited to this nerve alone is an unusual occurrence. It may, however, occur as the result of any form of local pressure or injury which alone involves this nerve trunk, and in order to affect all the filaments, it must occur in the region of the coracobrachialis.

Clinical Signs:—The sensory changes are limited to the lateral side of the forearm and extend from just below the bend of the elbow to the wrist since the musculocutaneous nerve becomes totally sensory at the bend of the elbow or just below it.

A complete paralysis of the nerve results in a total loss of power in the biceps and coracobrachialis and an incomplete paralysis of the brachialis muscle since it also receives a motor branch from the radial. As a result, the power to flex the forearm on the arm is greatly impaired, and would be totally lost if the brachioradialis and brachialis were not capable of assisting this movement.

Median Nerve. This nerve may be injured in its course through the arm by fractures or dislocations of the humerus, cuts, stabs and gun-shot wounds, but in civilian life is most frequently injured just above the wrist by glass wounds resulting from car accidents, broken bottles, etc., the resulting clinical phenomena will depend upon the site of the lesion.

Clinical Signs:—(a) If divided just above the wrist, there is loss of epicritic sensation over the palmar aspect of the radial side of the hand, as well as of the thumb, index, middle and lateral half of the ring fingers, and over varying portions of the dorsum of the same; loss of protopathic sensation over a much more limited portion, depending upon the cutaneous distribution of the ulnar and lateral cutaneous nerve of the forearm.

There is paralysis of the outer group of short thumb muscles (abductor pollicis brevis, opponens pollicis and flexor pollicis) as well as of the lateral two lumbricales. As a result, the hand assumes a most characteristic attitude; the thenar eminence is wasted and the patient is unable to oppose the thumb and fifth finger, the thumb remaining extended by the side of the fingers ("ape" hand). Due to the involvement of the lumbricales, flexion of the index and middle fingers at the metacarpal-phalangeal joints is weak.

(b) If division of the nerve occurs at the bend of the elbow or in the arm, to the above described symptoms are added the following; paralysis of the flexor group of muscles of the forearm, including flexor carpi radialis, pronator teres, pronator quadratus, brachioradialis, lateral half of flexor digitorum profundus, flexor digitorum sublimus, flexor pollicis longus and palmaris longus. As a result, the hand is strongly adducted, due to the unopposed action of the flexor carpi ulnaris when flexion of the wrist is attempted. The action of pronation is seriously impaired. The medial three fingers may be partially flexed since the fibres of flexor digitorum profundus which supply power to them are enervated by the ulnar. The middle phalanges of all the fingers and the terminal phalanges of the index and middle fingers cannot be flexed, but flexion of the proximal phalanx with extension of the other two can be performed in all fingers by the aid of the interossei which are supplied by the ulnar nerve.

Ulnar Nerve. Due to its superficial position at the elbow, where it lies in the groove between the olecranon and the medial condyle of the humerus, this nerve would be thought to be particularly prone to injury. It is, however, infrequently affected with traumatic paralysis. The causes which reported cases show to have produced this condition include the same type of accidents as were noted above for the median nerve. Poorly constructed crutches may also affect the nerve (crutch palsy) and in some cases, anatomical anomalies or cubitus valgus may be predisposing factors.

Clinical Signs:—As in the case of the median nerve, these will depend upon the location of the injury.

(a) If the nerve is divided at or above the elbow, there is a loss or impairment of sensation of the medial half of the ring and all of the small finger on the palmar side, a corresponding area on the palmar and dorsal aspects of the hand, and of the dorsum of the above fingers as far distally as the terminal phalanx (approx.).

There is a paralysis of flexor carpi ulnaris, and medial half of flexor digitorum profundus in the forearm group, plus all the interossei, the medial two lumbricales, the muscles of the hypothenar eminence, the adductor pollicis and palmaris brevis.

As a result, the hand is deformed into the "main en griffe" or claw hand—the extensor communis digitorum extends all the proximal phalanges of all the fingers while the other two rows of phalanges are flexed by the flexor digitorum sublimis (and part of the deep flexor). The fingers cannot be abducted or adducted, and the thumb cannot be adducted perfectly. If the paralysis has been of sufficient duration, there will be marked atrophy in the metacarpal (interossei) spaces as well as of the hypothenar eminence. Due to the paralysis of the one and one-half forearm muscles, attempted flexion of the wrist is imperfect and there is a weakness in the flexing power of the ring and small fingers.

(b) It will be apparent from the above that if the nerve is divided or injured distal to the point at which the branches to the forearm muscles come off the clinical features will be confined entirely to the hand.

Radial Nerve. This nerve is more frequently affected with paralysis than any other nerve of the upper limb. This may be largely attributed to the peculiarity of its course, and the fact that it lies in such close relationship to the shaft of the humerus in the spiral groove. It is damaged most frequently due to cuts, crushing injuries, fractures of the humerus, pressure due to sleeping on it, etc.

Clinical Signs:—If the nerve is divided in the upper one-third of the arm, that is above the origin of the external cutaneous branch, there is a loss of both epicritic and protopathic sensation over the radial half of the dorsum of the hand. Section of the nerve in the upper one-third of the forearm causes no loss of sensation in the forearm itself, while section in the lower one-third produces a limited loss of epicritic sensation over the back of the thumb.

Total division of the nerve is followed by paralysis of the extensor muscles of the forearm, the triceps and a weakening of the brachialis. As a result, there is a wrist drop or flexion, the fingers are flexed, as is also the thumb, the fingers usually being curled around it. When the patient attempts to extend the fingers, the interossei and lumbricales alone can be made to act, and these muscles, as has been mentioned before, can only extend the two more distal phalanges, their action on the first phalanx being one of flexion. The thumb and index finger cannot be extended or abducted, the patient cannot supinate the hand when the forearm is extended (this position being assumed to exclude the action of the biceps muscle) nor can the forearm be semi-flexed and the hand half supinated by the brachioradialis. When the patient is instructed to flex the forearm when placed in a position of half-flexion and semi-pronation, the brachioradialis lies flaccid and does not become tense and hard as in health.

The loss of power in the triceps makes extension of the forearm impossible when the arm is first raised above the head, nor

can the forearm be extended with the same degree of force in any position of the arm. When the hand is laid upon the table, the patient is unable to raise it from contact with the table, abduction and adduction of the fingers are, however, unimpaired. The action of the flexor muscles of the wrist seems feeble, since the lack of power in the opposing extensors fails to provide a fixed point of action. If, however, the wrist be forcibly extended and fixed by the examiner, it will be found that the flexors are still active.

Conclusions

It will be realized that the nerve lesions which have been described in this article are of the simple type, and that the effects described may vary with variations in the lesion, for example, section of one root of the median nerve will not produce all the symptoms that are associated with section of the trunk. Any combination of these may also occur:—A cut at the wrist may damage both the ulnar and median nerves—however, if an analysis is made of the functions of the hand, forearm and arm which are affected, a correct diagnosis of the nerve or nerves involved should not be too difficult. The affect on the sensory supply to the limb resulting from paralysis of the different nerves is variable. Those described in this paper are the ones which may be expected in the majority of cases. Too much importance, however, should not be placed upon cutaneous findings, since there are such wide variations, and in some cases, the area may be taken over by filaments of another nerve if the lesion has been of sufficient duration.

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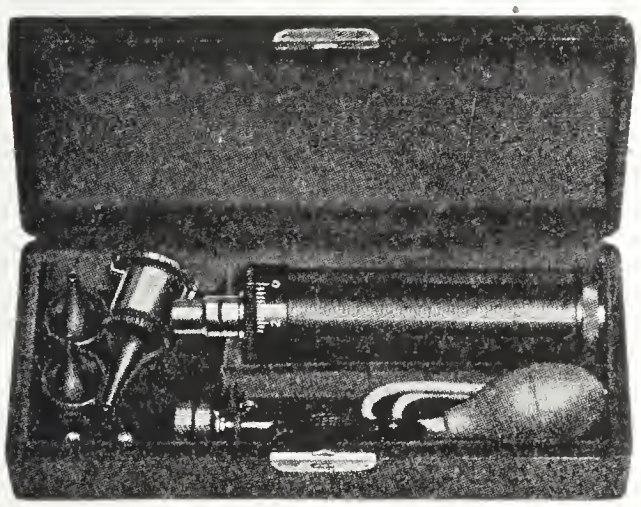

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THE MENSTRUAL CYCLE

By F. W. HANLEY, B. AND M., '40

Menstruation is defined by Bartelmez as *a bloody discharge associated with necrosis of the uterine mucosa, recurring, in the absence of pregnancy, at intervals of from three and one-half to five weeks in certain primates.* It has been observed in all human races and in all anthropoid apes and Old World monkeys which have been studied under favourable conditions. Actually, this periodic flow, evident at menstruation, is merely the outward manifestation of a cyclic process which occurs in the female reproductive organs.

The frequency and duration of the menstrual period and the amount of the discharge varies, both with the individual and with the age of the individual. Usually the periods are separated by about 28 days, plus or minus four to seven days; the flow normally persists for from three to seven days, although some variation from this may still be within physiological limits.

Sexual Cycle in Lower Animals

Most so-called lower animals, although they do not menstruate, exhibit periodically recurring sexual activity, known as rut, or oestrus. These periods are usually associated with a definite breeding season, and are correlated with certain changes in the reproductive organs. The animal first passes through a *pro-oestrus* stage in which there is increased vascularity and hypertrophy of the endometrium, proliferation of the vaginal mucous membrane, and desquamation of cornified cells from the walls of the vagina. At the same time, one or more of the ovarian follicles ripen, rupture, and discharge their ova. The ruptured follicles then form corpora lutea. In most of the mammalian species studied, there is no cyclic bleeding from the endometrium. However, in the bitch, the congestion is so great that the endometrium gives way in places, with the result that the whole pro-oestrus period, for a week or ten days, is marked by a definite bleeding which ceases about the time of ovulation. Also, in cows oestrus is sometimes accompanied by slight bleeding from the uterus. The phase of *oestrus* follows that of pro-oestrus. Coitus occurs in the former phase, in the majority of animals. The cornified cells of the vagina are removed by leucocytes, and the uterine glands show marked activity.

If oestrus is not followed by pregnancy, the uterus becomes more vascular, and there is hypertrophy of all the endometrial tissues. Since these changes are similar to those which occur when fertilization ordinarily takes place, this phase is known as the phase of *pseudo-pregnancy.* After the duration of a few days, necrosis replaces growth, and in a few mammals uterine hemorrhage actually occurs. Following this degenerative change, the endometrium returns to its resting condition. Thus, in mammals, there are two definite phases of the cycle at which bleeding occurs: in the pro-oestrus phase, due to the vascularity, and at the end of pseudo-pregnancy, due to necrosis of the endometrium.

Sexual Cycle in the Human

The human has no period of oestrus. Ovulation occurs about midway between successive menstrual periods. It is an interesting observation in this regard that menstruation among the Eskimos may not occur during the winter months. This may indicate a primitive breeding season in the human race.

The menstrual cycle in the human is not exactly comparable with the oestrus changes in other animals. The series of changes which occurs in the uterus is usually divided into four stages, although it must be remembered that such classification is merely for the purposes of description, the endometrium being in a state of almost continual change throughout the cycle:

(1) The *intermenstrual* or proliferative phase, with a duration of about 10 days,
(2) The *premenstrual* or secretory phase, with a duration of about 10 days,
(3) The *menstrual* or destructive phase, with a duration of about 5 days,
(4) The *postmenstrual* or regenerative phase, with a duration of about 4 days. Stages (4) and (1) are often included together, under the head of "repair or proliferative phase."

During the first phase, simple proliferation of the endometrium takes place, and many mitotic figures are observable in the epithelium and stroma. The glands increase in length and become more tortuous; vascularization increases, and the superficial capillaries become dilated. Blood may ooze into the uterine cavity at this time, producing one form of intermenstrual bleeding. Just before the end of this phase a mature ovum is discharged, and hence the phase corresponds to prooestrus. The proliferative endometrial changes are dependent upon the follicular or oestrogenic hormone, oestrin.

In the premenstrual or secretory phase, hypertrophy and secretion replace proliferation, hence mitotic figures can no longer be seen. The size and length of the glands increase, the glands become more tortuous and filled with secretion. Spherical translucent areas appear in the epithelial cells between the nucleus and basement membrane. These globules of secretion later pass to the free surface of the cell and escape into the lumen of the gland. The endometrium becomes still more vascular, and large thin-walled blood sinuses are formed, especially in the superficial layers. Blood escapes from these sinuses into the stroma. In addition, the whole endometrium becomes infiltrated with lymphocytes. Finally, the endometrium assumes a structure characteristic of the early stages of pregnancy; three zones can usually be recognized. In the superficial zone, the glands are straight and usually narrow and separated from one another by an edematous stroma. This zone may constitute one-quarter to one-half of the entire mucosa. In the intermediate zone, the glands are sinuous or sacculated, usually with widely dilated lumina. Finally, there is the basal layer, containing the bases of the glands. Here there is little secretory activity.

The secretory phase of the human cycle corresponds to the pseudo-pregnancy of certain animals. The changes described above are dependent upon the action of the hormone of the corpus luteum, which has been developing from the ruptured follicle. However, the uterus will respond to this hormone only if it has previously been under the influence of oestrin.

As pointed out by Meyer, the menstrual or destructive phase resembles an indirect circulatory necrosis. i.e. there is vasoconstriction with resulting ischemia of the endometrium, and subsequent relaxation of the vessels. The features of the menstruating mucosa are: extravasation of blood, necrobiosis superficially, leucocytic infiltration, and, after the first day at least, the gland cells are small and the stroma dense. This last is due to the dehydration of the mucosa. The necrosed tissue is shed into the uterine cavity along with erythrocytes and leucocytes. The extravasation may be slight or massive, and usually includes all the superficial layer and the bulk of the intermediate layer. By the third day, the shedding of the tissue is completed, so that the glands in the deeper portion of the spongy layer open directly into the cavity of the uterus. Bleeding continues from the capillaries of this layer.

Before the end of the menstruation, much of the denuded surface is again covered by epithelium which migrated radially from the lining of the glands to cover the exposed stroma.

During the postmenstrual or regenerative phase, the reparative processes continue. The superficial circulation again becomes active, and mitotic figures become abundant. As the glands resume their simple tubular shape and the stroma becomes readjusted, the lymphocytes withdraw from the latter. The cycle is now complete.

The Cause of the Menstrual Flow

In the light of experimental and clinical evidence, the most plausible theory

at the present time is that the intermediate control of the menstrual changes in the uterus depends upon the ovarian hormones, viz., oestrin and the hormone of the corpus luteum. These hormones, in turn, are controlled by the gonadotropic hormones secreted by the anterior lobe of the pituitary gland. As mentioned above, the proliferative changes of the endometrium progressing from the end of the postmenstrual phase throughout the intermenstrual phase are caused by the follicular hormone, oestrin, which apparently is secreted mainly by the tissues of the maturing Graafian follicle. By the time that the premenstrual phase has started, the corpus luteum has begun to develop from the Graafian follicle which shed its mature ovum at the end of the preceding phase. Hence the premenstrual phase is associated with both oestrin and corpus luteum hormone. It is the latter, according to Corner, which induces the typical premenstrual reaction of the endometrium. About two days before the end of the premenstrual phase, the corpus luteum begins to degenerate and there is a sudden cessation of the production of both the follicular and the corpus luteal hormones. Degeneration and bleeding of the endometrium follows (menstrual phase). If fertilization had occurred before the end of the premenstrual phase, the corpus luteum would have continued to develop and the endometrium would have passed from this phase to the typical decidual endometrium of pregnancy. This suggests that it is the sudden withdrawal of both ovarian hormones from the circulation which is responsible for the degenerative changes characteristic of the menstrual phase. In support of this, there is the evidence that complete removal of both ovaries (in either humans or monkeys) brings on a period of temporary uterine bleeding quite similar to menstruation. Allen found that this occurs even if there is no large follicle or corpus luteum in either ovary. This effect is prevented if injections of oestrin are given. A similar transitory bleeding occurs when oestrin treatments of spayed monkeys are suddenly stopped. Thus the conclusion is drawn that the bleeding is associated with a reduction in the oestrin level of the blood.

Extirpation of the corpus luteum at any stage of the premenstrual phase is almost invariably followed by menstruation. Merely dropping the extirpated corpus into the body cavity is sufficient to prevent the bleeding. However, the presence of a corpus luteum is dependent upon ovulation, and it is well established that in the monkey, between mating seasons, menstruation occurs without any preceding ovulation or associated uterine hypertrophy. These anovulatory cycles are quite normal both clinically and histologically. Anovulatory menstruation is also known to occur occasionally in the human. Thus, it appears that menstruation is possible without the formation of a corpus luteum and the typical premenstrual phase which it induces. On the other hand, oestrin and the proliferative changes which it induces in the endometrium are necessary for menstruation. From this the conclusion may be drawn that the corpus luteum and premenstrual endometrium, although normally associated with the menstrual cycle, are not essential for its occurrence.

Unfortunately, quantitative determinations of the amount of oestrin in the blood of the human during the menstrual cycle have not been adequate to show that there is an abrupt fall in level preceding the onset of menstruation. Even should this be proved, Cannon points out that, since there is no proof that the oestrin experiments are really specific in producing menstruation, the most that can be concluded at present is that such a premenstrual fall in blood oestrin is associated with the beginning of menstruation. That is, the oestrin reduction may be produced by the same conditions which bring about the characteristic vasoconstriction which initiates the menstrual phase.

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Abstracts

CRAWFORD B. SHIER, Editor

Contributors A. M. Crossland, F. L. Lawson, G. R. Walker, J. G. Watt

THE CHEMISTRY OF SULPHANILAMIDE AND SULFAPYRIDINE

C. C. LUCAS, M.A.Sc., PH.D.
Bulletin of The Academy of Medicine, Toronto, February, 1940

According to the descriptive nomenclature adopted by chemists, sulphanilamide is also known as paraminabenzene-sulphonamide. It is odourless, with a somewhat bitter taste, 0.8% solubility in water at room temperature, 1.5% solubility in water at body temperature; the aqueous solution is neutral to litmus. This chemical dye is much more soluble in alcohol and is very soluble in acetone; almost insoluble in ether, chloroform and benzene. The pure crystals melt at 166° C. Chemists have been able to modify the sulphanilamide molecule in three ways: (1) by replacing hydrogen atoms in the benzene nucleus by other atoms or groups, (2) by changing the amino group on the ring, or (3) by replacing hydrogen of the sulphonamide group.

Although several hundred derivatives have been prepared, only a few have shown any promise as anti-bacterial agents. While some derivatives of the (3) type are as active as sulphanilamide against streptococci, one of them, M. and B. 693, has become of great medical importance because of its anti-pneumococcal properties. It is also known by the chemists as 2-(p-aminobenzenesulphonyl)—aminopyridine. In the United States it is called sulfapyridine. It is of interest that while the range of activity has been broadened by this change in the molecule, there has been no loss of activity against the organisms affected by sulphanilamide. In fact, in-vitro tests indicate that it is more active than the parent substance against all organisms tested.

For parenteral use M. and B. 693 or Dagenan, being only sparingly soluble in water, is more soluble in the form of hydrochloride or a sodium salt, and this form of the drug is more useful for such a purpose. One liter of solution would have to be injected in order to give 1 gram of Dagenan, but a corresponding quantity of sodium salt can be dissolved in less than 4 c.c. of saline.

Both compounds, (sulphanilamide and dagenan) with an NH_2 group on a benzene ring are easily converted to intensely coloured dyes. This property has been utilized to determine colourimetrically their concentration in the blood and other body fluids.

Sulphanilamide is excreted by the kidney, partly in the acetylated form (therapeutically inactive). The concentration of the drug in the urine is usually about ten to twenty times that in the blood. Biochemical control is advised to obtain adequate dosage without toxicity.

C. B. S.

DECOMPRESSION IN THE TREATMENT OF INTESTINAL OBSTRUCTION

CHAS. C. JOHNSTON, M.D., M.S., F.A.C.S.
Detroit, Mich.

Decompression is the oldest and most obvious form of treatment for intestinal obstruction. In this article decompression is taken to mean the use of tubes in the intestine.

The underlying principles of various methods of decompression by means of intubation are the same; differences lie only in the level from which the material is aspirated. A method most applicable is to use a Miller-Abbott tube about ten

feet long or a cheaper double tube described. By getting the tube in the duodenum the balloon is inflated and peristalsis pushes it along.

The disadvantages are mainly two, (1) the possibility of failure to recognize interference with mesenteric blood supply—this is usually possible on the basis of history, physical examination, and reaction of the patient. (2) Difficulty of introducing the tube—in 3 years they had 6 failures and 500 successes.

The advantages are: (1) It offers a means of relieving the patient's obstruction without an operation. (2) It delays surgery until the patient is in better shape. (3) The operation can then be one of election and not emergency. (4) It helps to localize the lesion by means of little barium. (5) The patient can be fed above it and so avoid parenteral administration of fluids.

A. M. J. C.

SULPHANILAMIDE IN UROLOGY

D. R. MITCHELL, B.A., M.B., M.S., F.R.C.S.
Bulletin of The Academy of Medicine, Toronto, February, 1940

As result of study it was concluded, (1) that sulphanilamide as a urinary antiseptic can be used routinely in treatment of urinary infection; (2) that complications or toxic reactions were extremely rare on the scheme of dosage recommended in this paper; (3) that it cures more patients faster and more economically than any other antiseptic. Liver damage warrants increased vigilance for toxic effects, whereas severe kidney damage is a definite contra-indication to its use. Good effects with poor kidney function are not only difficult to obtain but practically impossible to maintain. Small dosage if effective produced sterile urine just as soon, (2 to 4 days) as the larger doses.

Concerning pyelitis of pregnancy, sulphanilamide was again found to be not only safe but powerful as a sterilizer of the urine, taking only about seven days to do its work. Older methods of treatment are less efficient. B. coli is usually the infecting organism. Pyuria in the puerperium is frequently due to or associated with pelvic inflammation. As the infecting organism may be a streptococcus, one does not expect such excellent effects as are obtained in prepartum pyelitis. Small dosage was also effective for treatment and must be carried on at the same level at which clearing of the urine occurs. The most suitable drug having antiseptic and least toxic properties is neoprontosil and, therefore, the most suitable drug in the treatment of urinary infections of kidney and bladder. All others have proved effective as urinary antiseptics, but some are too toxic. Dagenan is too toxic for routine use in the treatment of pyelitis and cystitis.

Dagenan in this present time is the agent of choice in the treatment of Gonorrhoea, because it acts more quickly than sulphanilamide, and gives reports of 80 to 100% cures.

C. B. S.

THE BLOOD-PRESSURE RAISING SECRETION OF THE ISCHAEMIC KIDNEY

By FASCIOLO, J. C., HOUSSAY, B. A., AND TAQUINI, A. C.
J. Physiol. 1938, 94: 281-293

Renal ischaemia and permanent hypertension with 50-100 mm. Hg rise above the initial blood pressure were produced in 185 dogs by compressing the renal artery and reducing its calibre by means of an adjustable forceps. The ischaemia of the kidney is the cause of the hypertension, since on removal of an unilaterally treated kidney the raised blood pressure rapidly falls to normal. The hypertension is not due to uraemia, since in many animals this is absent. This experimental hypertension due to renal ischaemia is produced through a humoral and not a nervous mechanism, since it can be produced and maintained after large resections of the nervous vasoconstrictor system.

Extracts of kidneys from dogs with hypertension caused a larger increase in blood pressure than extracts of kidneys from normal dogs. By grafting ischaemic

kidneys into the necks of chloralosed, recently nephrectomized dogs it was shown that the ischaemic kidney induces hypertension by producing a secretion which causes a rise of the normal blood pressure till it reaches hypertension level and maintains it there. In addition, by means of profusion with venous blood plasma it was shown that the ischaemic kidney produces a substance with an intense direct vasoconstrictor action, and that the ischaemic kidney is the essential or only site for the production of this substance in the dog with hypertension. Normal healthy renal tissue eliminates or destroys in varying degree the vascoconstrictor substance, reducing the rise in blood pressure produced by an ischaemic kidney. The vasoconstrictor substance is active in the absence of thyroid and adrenal glands.

F. L. L.

THE SEVERER FORMS OF APPENDICITIS WITH SPECIAL REFERENCE TO THE TREATMENT OF APPENDICEAL ABSCESS

ERNEST E. ARNHEIM, M.D., F.A.C.S., AND HAROLD NEUHOF, M.D., F.A.C.S.
New York
Surg. Gync. and Obst. Jan. 1940

This paper is based on an analysis of the personal experiences of the authors. They are convinced that death should rarely result from acute phlegmonous or gangrenous appendicitis, appendicitis with local peritonitis or appendicitis with abscess.

The authors dislike attempts to produce rebound tenderness, looking upon it as an unduly rough manoeuvre which might spread infection and which is often elicited in the absence of peritonitis. They do not operate on admission in acutely ill, dehydrated patients who have been sick two days or longer. These patients are observed continuously and are given several hours of supportive treatment such as intravenous infusion before operation. An exception is made in young children and infants since there is small tendency for the walling off of peritoneal infection.

They believe it safer to defer operation in appendiceal abscess until the inflammatory process has subsided in part. Intravenous fluids, and bland fluids by mouth are given. Enemata and frequent abdominal examination are interdicted.

They do not use the routine McBurney but have special incisions for their particular requirements, and the site is placed over the assumed position of the lesion. The appendix is released and delivered by a "no touch" technique that they devised.

Drainage is done for (1) a localized collection of frankly purulent exudate. (2) Purulent exudate extending beyond the limits of the appendix. (3) An infected appendiceal bed. (4) A doubtful appendiceal stump.

Intravenous glucose is started before, carried through, and maintained after operation in the seriously ill. Adequate opiates are given for rest. No cathartics are used. They take a conservative attitude as regards to the indications for operative interference for postoperative intestinal obstruction, believing that the fibrinous exudate that is responsible for the angulation of the gut will be absorbed thus releasing the obstruction. Postoperative complications were pneumonia, pelvic abscess, paralytic ileus, intestinal fistula, acute cholecystitis, auricular fibrillation, respiratory depression due to avertin. There were no postoperative hernias.

J. G. W.

THE INTRAVENOUS INJECTION OF SODIUM SALTS IN SCIATICA

H. B. SUTTON
Lancet 327: 1168-1169 Dec. '39

The injection of sodium salicylate and sodium iodide—15 grains each in a single solution of 20 cc. water in 32 cases of sciatica are reported. The injection was made slowly into a vein in the elbow and care was exercised to prevent entrance of the solution into the tissues as this causes considerable pain.

In cases of primary sciatica (20) the sciatic pain usually ceased in about ten minutes after the injection had been completed. In some cases it returned in 18

hours and a second injection brought better results.

It is evident that a solution of sodium salicylate and sodium iodide given intravenuously will rapidly cure uncomplicated cases of primary sciatica.

On the other hand, in the cases of sciatica due to some extraneous pathological condition (12) the injection diminished the pain about 3 hours only and a second injection was even less effective. The results of the injection, therefore, may be used to differentiate primary from secondary sciatica.

A. M. J. C.

PLASMA POTASSIUM FOLLOWING INTESTINAL OBSTRUCTION IN DOGS

W. F. GREENWOOD, R. E. HAIST, AND N. B. TAYLOR, *Toronto*
Surgery, Feb. 1940

Scudder and others have reported that in cats there is a rise in blood potassium to lethal levels following obstruction of the intestine at various points, and suggested that potassium is the toxic factor in acute intestinal obstruction. In humans, in 20 untreated cases of intestinal obstruction the plasma potassium was raised in seven and lowered in five and the conclusion was drawn that these disturbances explain some of the symptoms in intestinal obstruction.

The authors followed the serum potassium in twelve dogs with intestinal obstruction. In most dogs the obstruction was made about 2″ below the opening of the common bile duct. All showed a fall in plasma potassium in the first few days after operation, and a rise before death but only in one case above the pre-operative level. It is possible that there are terminal rises in the last few hours, but it does not seem logical that so late a rise could be of any significance.

Summary:—These observations indicate that in dogs changes in plasma potassium do not play an important part in causing death following acute intestinal obstruction.

G. R. W.

Book Review

THE PRINCIPLES AND PRACTICE OF AVIATION MEDICINE

By HARRY G. ARMSTRONG, B.S., M.D.
Director, Aero Medical Research Laboratory, United States Army
The Williams and Wilkins Co., 1939, 489 pages, $7.15

At a time when the Russians are harassing Finland by air; when the Germans are preying on North Sea shipping, and the Allies are reconnoitering far behind the Siegfried Line, one feels that this recent publication on Aviation Medicine is very appropriate . . . More appropriate still for Canadians, because Canada will soon play an extremely important part in the Empire Air Training scheme.

With all this aerial activity going on around us we cannot but be tremendously interested in that branch of Medicine which is inherently bound up with its welfare. This new science is presented as a distinct and separate subject and not as a disconnected collection of various other medical specialties. The book adequately prepares physicians that they may intelligently examine and advise those interested in aviation. It includes such subjects as selection and care of fliers, methods of examination, the etiology, symptomatology and treatment of common disorders resulting from flight, and the recent advances in flight safety. It is presented in a clearcut, simple and interesting manner by a scientist highly qualified to write on the subject; a book which deserves a place on everyone's library shelf.

E. B. TOVEE, M.D.

Feature Section

F. L. LAWSON, Editor

PROBLEMS IN AVIATION MEDICINE

By S. C. BEST, B.A., '43

With the year 1862 the world witnessed the first real success of man in his attempt to reach the clouds, when Coxwell and Glaisher ascended in a balloon to the height of 29,000 feet. But for the presence of mind of Coxwell, who grabbed the valve rope with his teeth, they would have made little contribution to physiological history. Glaisher noted loss of visual acuity and hearing, paralysis of legs and arms, and final unconsciousness.

Paul Bert, a French physiologist, heard of this work and started investigations on the effects of high and low pressures on man. In 1865 he sent three men up who reached 28,820 feet, but only one lived to tell the tale.

Three years after the turn of this century the Wright brothers achieved their first successful flight at Kitty Hawk, North Carolina, and powered aviation had its inception really at that time.

About 1916 the use of planes as war weapons was realized but, following the armistice, interest remained at a low ebb until the original work of Myers on the physiology of blind flight. Since then the field of aviation and aviation medicine has broadened and deepened rapidly.

The problems in aviation medicine may be generally classified according to their fundamental causes.

1. *The effects of acceleration*—linear, centrifugal, and angular; their amount, duration, and repetition.

2. *The effects of altitude*—actual altitude attained, duration of maintenance of any particular altitude, rate of change of altitude, and perhaps the factors of cold and light.

3. *The effects of vibration*—noise, sound, sound, and inaudible vibrations.

4. *The effects of noxious fluids, gases, strong winds*, etc.

5. *The effects of flying on the personality*—neuroses, etc.

Effects of Acceleration

A. *Linear and centrifugal accelerations.* These have been well termed the stress-producing accelerations. Speed as such is not important—we must remember that the average individual is constantly travelling some $18\frac{1}{2}$ miles per second due to the rotation of the earth. Acceleration may be defined as the rate of change of velocity. The acceleration due to gravity is taken as 1 G. The highest accelerations in present-day aviation are due to changes of direction of flight due to the positive or negative lift of the wings. Usually the force is exerted in the long axis of the flyer and perpendicular to the axis of the plane.

An upward acceleration is termed a positive acceleration, and downward a negative acceleration. A dive at 175 m.p.h. gives an acceleration of 9.3 G. In the long axis of the body, an acceleration of plus 5 G causes a temporary loss of vision due to draining of the blood away from the head. Five to 7 G results in coma. Due to a lag in the blood flow, 5 seconds of acceleration is required to obtain the full physiological effect. There is a wide variation between individuals in their vascular tone and vasomotor control, so there is considerable variation in the amount of acceleration that different people can tolerate. Rapidly repeated exposures decrease tolerance, while daily exposures increase it. Tensing the abdominal muscles helps moderately. A broad belt exerting 100 mm. Hg. pressure helps markedly at the end of 30 seconds after application. Plus 12.2 G in flight and 16 G in the laboratory have been tolerated with no apparent injury.

Negative accelerations drive the blood up to the head. Minus 3 G results in the symptoms of concussion, and minus 5 G results in massive haemorrhage (in animals) in the cranium due to rupture of one or both of the posterior communicating arteries in the circle of Willis. With negative acceleration the latent period is brief, possibly about 1 second. There is no obvious means for counteracting the effects of the acceleration.

Accelerations in the transverse axis produce little serious result. 8 G causes compression of the supporting framework of the lungs and induces forced respiration, which is normally counteracted by involuntary holding of the breath in the midinspiratory position. The main problem here seems to be a matter of supporting the body for any anticipated direction of transverse acceleration.

B. *Angular acceleration.* Normal individuals suffer an illusion of reversal of motion during acceleration of vertical or rotary motion while deaf mutes with below normal or absent perception usually do not. Normals are more accurate than deafmutes in detecting tilts, banks, and turns in flight when blindfolded. Normal pilots coming out of a spin feel a reversal of motion and therefore frequently fall into a second, usually fatal spin. Vertigo can be avoided by disregarding the senses and flying by the instruments. Vertigo results from conflicting sensory impressions, and from loss of equilibrium or the fear of falling. It may usually be inhibited by visual orientation. It is frequently accompanied by manifestations of fear—pallor, sweating, muscular weakness, despair, etc. Airsickness is a form of vertigo, and varies with the amount and duration of acceleration.

Aerial equilibrium is maintained by the eyes, the ears, deep sensibility, the viscera, and the skin in this approximate order of importance. The ear informs of direction and the deep sensibility informs of movement. For years aviators have recognized what they call *flying by the seat of the pants*—a combination of deep sensibility, visceral sensation, touch, pressure and other skin sensations.

Effects of Altitude

The primary effect of altitude is of course that of anoxia. The term refers to deficiency of oxygen in the body tissues. Glaisher in 1862 had noted loss of visual acuity, paralysis, etc., with altitude. Bert had shown that the effect was due to a decreased oxygen partial pressure. The percentage composition of the air remains practically constant up to at least 72,000 feet, but with increased altitude the pressure decreases, the pressure of oxygen always amounting to about one-fifth of the total. The atmospheric pressure at which the alveolar partial pressure of oxygen reaches zero is not 47 mm. Hg. (62,965 feet) as is usually stated but at 86 mm. Hg. (50,313 feet)—an altitude amost 13,000 feet below the generally accepted level. Breathing pure oxygen instead of atmospheric air does not increase the altitude at which the alveolar oxygen tension in the lungs reaches zero but this condition is reached at the same altitude in both instances. Thus there is a very definite altitude above which it is impossible to ascend while using oxygen. There is little doubt that before long, pressure cabins will be in common use. With anoxia in the body the respiratory centre is weakly stimulated and lung ventilation is increased, so washing out more carbon dioxide. Since carbon dioxide is a powerful respiratory regulator, this washing out checks the increase in respiration until a new rate is established. At first there is also an increase of pulse rate and blood pressure, but in time the normal state is resumed for reasons that are still not apparent. Also the Bohr effect comes into play with anoxia—when the carbon dioxide of the blood is below normal as a result of increased lung ventilation, the blood has a greater affinity for oxygen and so becomes more highly saturated as it passes through the lungs. But the blood does not release its oxygen to the tissues, the net result being a greater tissue anoxia.

Airplanes have attained an altitude of 56,016 feet and stratosphere balloons 72,395 feet. The average modern plane has a ceiling of about 20,000 to 30,000

feet, but most long commercial runs are at 8,000 to 14,000 feet. The tolerable altitude varies with the rate of ascent. The more rapid the rate of ascent, the greater the effect on the central nervous system and the slower the ascent the greater the effect on the cardiovascular system. A rate of ascent of 500 feet per minute is the optimum for endurance of anoxia over a period of time. The faster rate prohibits use of immediate compensatory mechanisms, and the slower rates subject the body to accumulative effects of anoxia for considerable periods. It requires 24 to 48 hours to recover from a moderate anoxia of a few hours, or from an extreme anoxia lasting but a few minutes. The duration of exposure to an altiude of 25,000 feet matters little unless prolonged to about 25 minutes, at which time there is a paralysis of the respiratory centre followed by death. Altitude tolerance varies with inherent tolerance, physical activity, general physical condition, the emotional state, and clinical anomalies.

Altitude sickness is the result of anoxia. The first symptoms appear at 9,000 feet, with unconsciousness at 25,000 feet at a rate of ascent of 1,000 feet per minute. Flying at 10,000 to 12,000 feet for two to four hours results in a dull headache and a sense of fatigue which may persist for hours. Exposure to 15,000 to 18,000 feet for two to six hours may result in nausea, vomiting, mental confusion, muscular weakness, etc. Death, when it occurs, is probably due to failure of the respiratory centre.

Altitude exposures cause a decrease in hemoglobin and red cell count and an increase in the white cell count of the blood.

Hearing starts to decrease at 16,000 to 18,000 feet and may be abolished at 20,000 feet. Vision behaves similarly but may be present until coma supervenes. From 14,000 feet up there is a progressive deterioration of voluntary muscular control.

Chronic altitude sickness results from repeated exposures to high altitudes. It is marked by increased lassitude, excess (unrefreshing) sleep and hypertrophy, with later degeneration of the adrenals. Cure of the condition is obtained by cessation of exposure and may require from 48 hours to two months.

Effects of Vibration

The effect of noise is decreasing in aviation due to more adequate insulation, etc. The effect of vibration seems to be largely a matter of fatigue, both mental and physical. It has been suggested that this is due to a constant disequilibrium and the very basic fear of falling which may be subconscious. The skeletal muscles become tense, and the effect is increased if the absorbing area is small.

Effects of Noxious Fluids, Etc.

The effects of gases, fluids, wind, heat, etc., are more readily controlled and are fairly obvious.

Effects on Personality

Due to the strain on the nervous system as well as the physique, any abnormalities of the personality are usually enhanced. Requirements for flying may well include good physique, youth, intelligence and education, fair athletic ability, moderation with tobacco and alcohol, and healthy temperament.

Conclusion

The field of aviation medicine is developing very rapidly and has particular significance at this time. The interested reader would be well advised to read H. G. Armstrong's book "Principles and Practice of Aviation Medicine," 1939.

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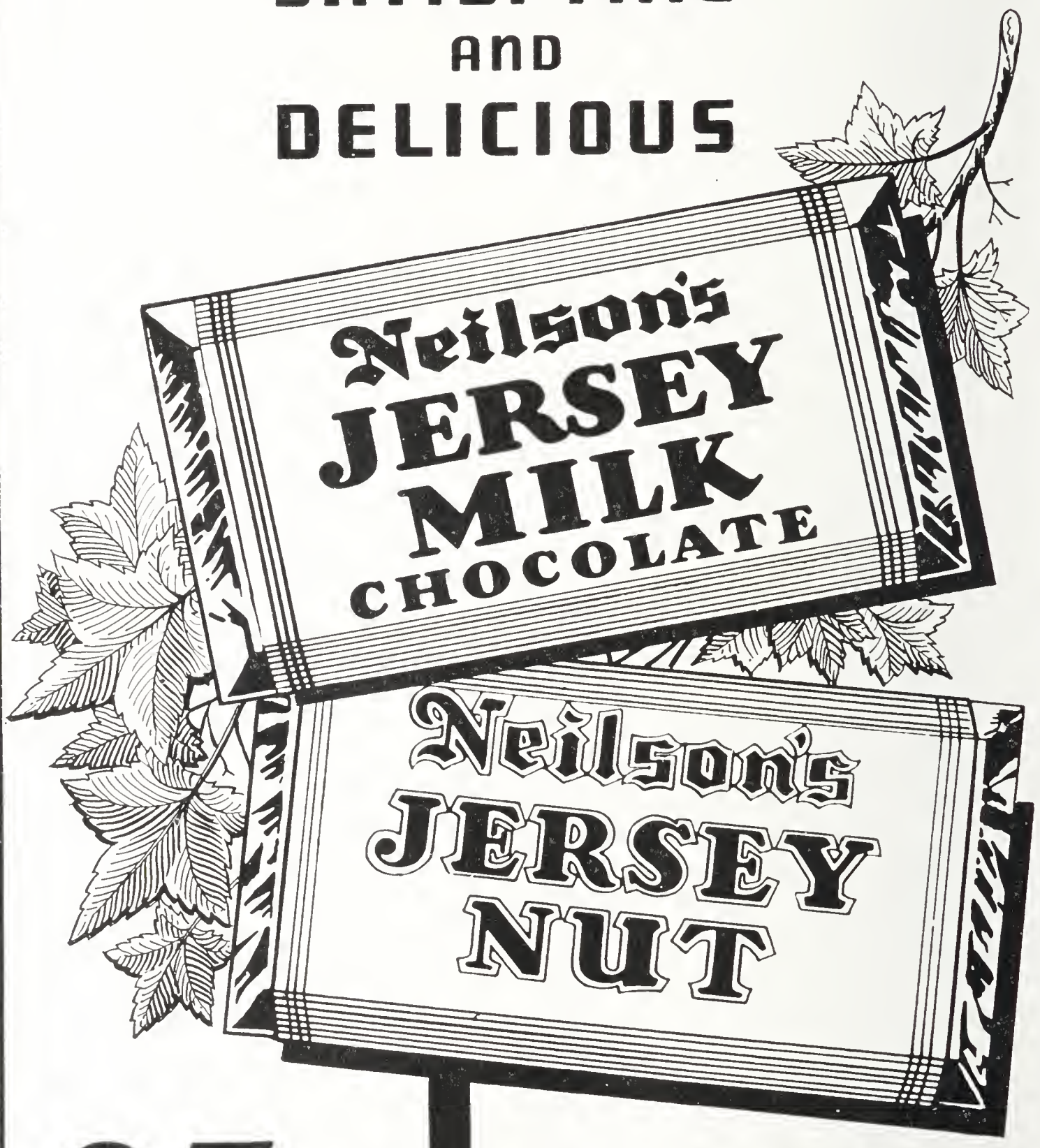

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C.A.M.S.I. Notes

By A. J. DENNE

In early March, the annual C.A.M.S.I. convention will be held at Queen's University, Kingston, Ontario. The 1940 convention will be the third in the history of C.A.M.S.I. and we hope it will prove the value of such an organization to every medical undergraduate in Canada.

In the April issue of this Journal, we plan to publish the reports of the convention delegates from the various medical schools in Canada together with the accomplishments of the Association to date and plans for the future. The reports, for obvious reasons, will be presented in both French and English. This final issue will be distributed to every medical student and interne in Canada and should promote a greater national interest in C.A.M.S.I. affairs.

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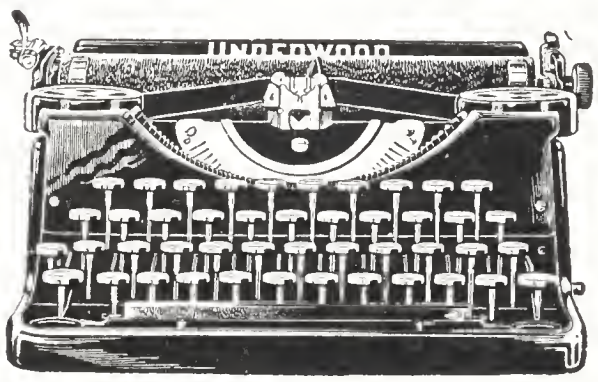

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MEDICAL SOCIETY, UNIVERSITY OF TORONTO

At an Open Meeting of the Medical Society on Monday, February 12, an audited account of the transactions of the Medical Society for the academic year 1938-39 was requested. It is with pleasure that we publish this account which will be discussed at a date to be announced in the near future.

STATEMENT OF RECEIPTS AND DISBURSEMENTS AND INCOME AND EXPENDITURE
For the period July 29th, 1938 to October 31st, 1939

	RECEIPTS	INCOME
Cash on hand—July 29th, 1938	$ 424.19	$ 424.19
Fees from Bursar during above period	3,100.00	3,100.00
Amounts transferred from:		
Daffydil Committee—1937-38	132.45	132.45
" " —1938-39	.....	197.24
Medical At Home Committee—1937-38	402.16	402.16
Medical Athletic Association—1938-39	.....	13.21
TOTAL RECEIPTS AND INCOME	4,058.80	4,269.25

	DISBURSEMENTS	EXPENDITURE
Undergraduate Women's Medical Association	$ 171.00	$ 171.00
Medical Athletic Association	1,325.00	1,325.00
Class of 1939 Meds U. of T. graduation banquet	100.00	100.00
Canadian Association of Medical Students and Internes	56.75	56.75
Bursary Committee—Faculty of Medicine	.....	300.00
Medical Journal—fees	796.00	796.00
Christmas donations to employees	61.66	61.66
Medical At Home Committee	500.00	500.00
Medical At Home tickets	70.00	70.00
Daffydil Committee	500.00	500.00
Daffydil tickets	20.00	20.00
Soph Frosh—deficit on dance	55.00	55.00
" " —unpaid account to Royal York Hotel	.....	20.00
" " —tickets	32.50	32.50
S.A.C. Torontonensis	25.00	25.00
Miscellaneous (see attached list)	302.51	341.22
TOTAL DISBURSEMENTS AND EXPENDITURE	3,665.42	4,024.13
Balance of monies on hand represented by cash in bank as at October 31st, 1939	393.38	.....
Excess of income over expenditure for the period ended October 31st, 1939	.....	245.12
TOTALS	$4,058.80	$4,269.25

MISCELLANEOUS DISBURSEMENTS AND EXPENDITURES

	DISBURSEMENTS	EXPENDITURES
Telephone	$ 14.55	$ 14.55
Caretaker	4.25	4.25
Stationery and printing	46.79	46.79
Medical Society Keys	51.14	52.85
Travelling expenses for Paul McGoey to Queen's University	34.00	34.00
Travelling expenses for B. Laski to O.A.C. Guelph	26.50	26.50
Travelling expenses for R. S. Braiden to London	23.25	23.25
Travelling expenses for C. C. Gray to Montreal	58.50	58.50
Photographs	42.50	42.50
Audit expense for period July 29th, 1938 to October 31st, 1939	.....	20.00
Stamps and sundries	1.03	1.03
	$302.51	$341.22

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• • •

(1) Dick, Geo. F.: The Yearbook of General Medicine 1939, p. 92. (The Yearbook Publishers Inc. Chicago).
(2) Kendrick, P., and Eldering, G.: Am. J. Hyg., Sect. B, **29**:133 (May) 1939.
(3) Sauer, R. W.: J.A.M.A. **112**:305 (Jan. 28) 1939.

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